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MANAGEMENT OF POSTOPERATIVE SUBTOTAL GASTRECTOMY PATIENTS *

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The treatment of intractable peptic ulcer or complicated peptic ulcer is best accomplished by subtotal gastrectomy. The procedure is not without sequelae and the end results of the operation should be carefully considered before such surgery is recommended to the patient. Most patients who have such surgery are cured of their peptic ulcer. The exact percentage of cures is difficult to evaluate since objective study, including radiologic and endoscopic examination of the patients is not accurate. In addition to the recurrences of peptic ulcer following subtotal gastrectomy a significant number of patients develop new symptoms as a result of surgical procedure. The symptoms following removal of part of the stomach may be mild or severe. Various authors differ in their evaluation of poor results following gastrectomy for peptic ulcer. The severity of the symptoms prior to surgery and the reluctance of the surgeon to operate for mild disease will affect the results. The patient who has serious disease before surgical intervention is recommended will have the best chance to obtain benefit. In my experience there have been many patients who have had moderate to severe postgastrectomy symptoms who have been happy with the results of surgery since their ulcer was more disabling than the postgastrectomy symptoms. Other patients with rather mild postgastrectomy complaints were unhappy because of relatively mild symptoms following surgery since their original disease was relatively mild.

In the preparation of this paper the records of 51 private patients were reviewed. These patients all had moderately to severe disabling postgastrectomy complaints. The purpose of this paper is to review some of the theoretical considerations in the production of the postgastrectomy syndrome and to correlate the theoretical considerations with clinical experiences with patients.

A single term to describe the situations that occur following surgery has not yet been coined. The expression "dumping syndrome" has been widely used. The dumping syndrome probably represents only one phase of the clinical picture that can occur following removal of a large portion of the stomach. A discussion of the changes that may occur following gastrectomy involves the consideration of many factors affecting the feeling of well being of the patient.

NUTRITION

The patients in this series have shown an average weight loss of approximately 20 pounds in relation to preoperative

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weight. The greatest loss in any individual was 80 pounds. The most favorable results from the standpoint of nutrition have been in those patients who have been somewhat overweight at the time of surgery. The poorest results have been in those patients who, at the time of surgery, were markedly underweight. Most patients who were underweight have not been able to gain weight postoperatively. Malnutrition of a marked degree was a problem in 10 patients. Eight of these malnourished patients were underweight when the surgery was performed.

Rapid transit through the gastrointestinal tract may be an important cause of the nutritional problem and may be accompanied by steatorrhea. Wollager,[1] in 1949, showed that the patients that he studied following subtotal gastrectomy excreted an abnormal percentage of fat regardless of the type of diet. Muir,[2] in 1949, found that 90 per cent of his patients had difficulty in gaining weight following surgery for ulcer.

Rapid emptying of the stomach and failure of proper mixing of the digestive secretions with the food bolus have been considered to be important factors in causing steatorrhea. Wells and Welbourn,[3] in 1951, found that the use of hexamethonium bromide slowed transit time and increased fat absorption. The inability to gain weight in some patients has also been attributed to the fact that the afferent loop may become filled with bile and pancreatic secretions and empty only at intervals. This filling of the afferent loop may cause discomfort after eating and because of this discomfort the patients are fearful of taking a normal intake at any one meal.

ANEMIA

Anemia is seen following postgastrectomy in some patients. The anemia is usually microcytic in type and responds well to administration of iron. The incidence of the postgastrectomy microcytic type of anemia is sufficiently common that some physicians recommend intermittent administration of iron routinely to the postgastrectomy patient. The secondary anemia is probably due to decreased absorption which may be influenced by the lack of HCl, and increased bacterial content of the small bowel. The use of hydrochloric acid or elements of the vitamin B-complex may be healthy adjuncts to the use of iron in treating the anemia seen following subtotal gastrectomy. Primary anemia is rare following subtotal gastrectomy but does sometimes occur. In total gastrectomies a primary anemia is certain to develop and usually occurs within two to four years. In our patients, there were only 8 of the 51 with a significant anemia. Anemias are usually not a serious problem since the response to simple therapy is adequate.

POSTCIBAL DISTRESS

Distress that occurs immediately following meals has given rise to the term dumping syndrome. Dumping was originally a radiological term. The exact meaning of the dumping syndrome is not clear. Frequently, both the early and the late symptoms following meals are associated with the term dumping syndrome.

For clarification of the symptoms and abnormal physiology, postcibal distress may be divided into three groups: first, the early complaints which occur the first sixty to ninety minutes after meals and are usually mechanical in origin. Second, the late symptoms occurring from one and one-half to two and one-half hours after meals usually of chemical origin and are associated with hypoglycemia. Third, the so-called "afferent loop syndrome" or "bilious vomiting" which creates increasing discomfort over a period of days and is probably due to progressive distention of the afferent loop. These three groups will be considered separately.

EARLY POSTCIBAL SYMPTOMS

The occurrence of the early postcibal distress has been attributed to mechanical factors. Rapid emptying of the gastric remnant and jejunal filling with the bolus of food may be a cause of discomfort. A similar type of distress may also occur

with edema or other obstruction of the stoma leading to marked delay in emptying of the gastric remnant. The symptoms which arise immediately after a meal usually last from fifteen to forty-five minutes. The common symptoms are a feeling of epigastric fullness, nausea, weakness, gas, palpitation, sweating and sometimes a feeling of faintness. These symptoms are often relieved if the patient lies down and are aggravated by the administration of more food. Glaessner, in 1910,[4] noted that there was nearly always hyperglycemia coincident with the early postcibal distress.

The physiological basis of the complaints is not clear but there have been many theories proposed to explain these symptoms. Machella,[5] in 1947, was able to demonstrate that jejunal distention could produce a typical early postcibal disturbance. He was able to produce this discomfort by the introduction of hypotonic solutions into the jejunum of postoperative patients as well as of normal individuals. He felt that the osmotic effect of the hypotonic solutions drawing fluids into the jejunum produced a mechanical distention which automatically produced the discomfort seen in the postgastrectomy patient. Butler and Capper,[6] in 1951, were able to produce the typical postcibal symptoms in 79 patients by distention of the jejunum and they also were able to produce the symptoms by torsion of the stomach stump by placing a mercury filled bag in the gastric remnant. The discomfort would be more noticeable when the patient was in the erect position. The swallowing of barium may also produce the early symptoms of the postgastrectomy syndrome. Capper,[7] in 1952, demonstrated that a gastric stoma may descend an average of 7 cm. on changing from the supine to the erect positions. If one assumes that the gastroesophageal junction is a fixed point then this change of position represents stretching of the stomach. The distress that can be produced by a mercury weight in the gastric remnant will always be relieved by having the patient lie down. The same amount of mercury does not produce symptoms in control patients who have not had gastrectomies. Splanchnic block relieved the symptoms in 77 per cent of the patients with symptoms caused by the mercury weight in the gastric remnant. Splanchnic block tried in 61 patients with the early postgastrectomy syndrome produced relief in 46 patients for from two to nine days and 4 of the 61 cases received complete relief by the block. The distress produced by distention of a balloon in the jejunum is unaffected by posture.

The above observations led Capper to postulate that the anatomy of the postgastrectomy stomach may play an important part in the production of symptoms. Normally, the stomach is fixed at its extremities and the lesser omentum supports the lesser curvature by way of the thickening that occurs around the left gastric vessels. Following high gastric resections the gastric remnant is unsupported. This unsupported remnant has the anastomosis hanging from it with the U-shaped afferent loop. The afferent loop may be weighted down with digestive juices and possibly reflux of food as well. It has been demonstrated under local anesthetic that traction on the stomach will produce the feeling of distention, nausea, and retching. Splanchnic block abolishes the above symptoms. Traction on the vagus does not have a similar effect. It is felt therefore that the syndrome is not mediated via the vagus nerve. Jordon, Overton and Debakey[8] have presented a different theory related to the dumping syndrome. They noticed that the chief symptoms experienced either singly or in combination were increased pulse, weakness, nausea, sweating, vomiting, vertigo, diarrhea, and abdominal cramps. They carefully studied 20 patients, 5 of whom had intestinal continuity re-established by gastroduodenostomy, and 15 by gastrojejunostomy. They placed a balloon 12 inches beyond the gastrointestinal stoma. They found that when the balloon was distended the first symptom produced

was pain. This distention did not produce the usual postcibal syndrome in any of the patients. They then injected hypertonic solutions and found that they could always produce the postcibal complaints by the administration of the hypertonic glucose solution in the jejunum and also by a test meal containing fat, protein, and a hypertonic glucose material. A protein solution alone did not produce complaints in most of the patients. They were able to show that the patients who developed symptoms showed varying degrees of hypermotility and that in symptomatic patients the use of antispasmodics produced an irregularly beneficial effect. They found that partial or complete relief of symptoms occurred in 81 per cent of the patients in whom they could demonstrate that drugs inhibited motility by 50 per cent or more. They therefore postulated that hypermotility is important in the production of postcibal complaints of nausea, vomiting, and diarrhea.

Kleiman and Grant,[9] postulated that low potassium levels caused early postcibal complaints. They point to the fact that ingestion of food, rich in sugar, is the most common trigger to the syndrome. They noted that the attacks frequently occur during the hyperglycemic period and have noted associated electrocardiographic changes—notably shortening of the QT interval and depressed T waves which they interpreted as consistent with hypopotassemia. The suggestion is made that the dumping attack is caused by the sudden shift of potassium from the extracellular fluid into storage in the liver with glucose. The authors were unable to demonstrate serum potassium changes but did show that 10 patients who were given 10 grains of potassium chloride twenty minutes before meals showed marked improvement. Smith, et. al.,[10] also studied the postprandial attacks of palpitation and weakness in relation to potassium. They were not able to demonstrate a direct relationship to potassium from the standpoint of the early complaints but noticed that the weakness following the attacks was greatly diminished if potassium chloride was given orally and more so if given intravenously. Muncke [11] and Roberts [12] studied postgastrectomy symptoms also in relation to potassium. Muncke did intravenous glucose tolerance tests on the patients and also used hypertonic glucose administered by mouth. During the oral glucose tolerance test there was no alteration in the concentration of serum potassium and the serum potassium did not vary in relation to the attacks. He did notice, however, electrocardiographic changes similar to those described by Smith and Kleiman. He stressed the difficulty of distinguishing electrocardiographically between increased adrenalin concentration and a hypopotassemia. Muncke stated that considering the various aspects of the clinical picture that the rise in blood pressure, the tachycardia and the vasomotor symptoms made it seem reasonable to attribute the changes to an increased sympathetic tonus. Intravenous glucose tolerance tests indicated that the intermediate carbohydrate metabolism is normal. He felt that attacks were due to increased sympathetic tonus and this was stimulated by chemical agents, particularly hypertonic glucose.

Roberts, Randall and Farr,[13] also studying the changes in electrocardiograms, electrolytes, and the use of hypertonic glucose solutions found that the blood volume was markedly diminished after oral administration of hypertonic glucose solutions to patients after gastrectomy and they felt that the drop in blood volume was a causative factor in the symptoms of the dumping syndrome. Their findings showed that patients with intact stomachs did not display the above described physiological alterations indicating that the solutions did not enter the intestine with the rapidity with which they did in gastrectomized stomachs. The well-known function of the stomach as a gastric reservoir provides the protection against rapid dilution or breakdown of food stuffs.

LATE POSTGASTRECTOMY COMPLAINTS

The late postgastrectomy symptoms usu-

ally occur two to three hours after a meal. In a typical day, the distress may occur at 11 A.M., and 3 P.M. and is more likely to occur if the patient is quite active. The symptoms consist of tremor, giddiness, profuse perspiration. There may be marked anxiety and exhaustion. Nausea and hunger may be present with a feeling of emptiness in the abdomen. In the severe cases, marked palpitation, faintness and even loss of consciousness may occur. The physical findings consist of tachycardia, fall in blood pressure, pallor, sweating and tremor. Glycosuria and diuresis may occur and in most instances hypoglycemia is present. This secondary hypoglycemic reaction usually disappears in two to five years following surgery. There have been many theories as to the cause of this hypoglycemia and accompanying symptoms. Zollinger [14] in 1947, noted that when these patients developed weakness and sweating two hours after meals they almost always had a hypoglycemia. In 1942, Evensen,[15] observed that hypoglycemia disappeared in a very significant number of patients if the gastrojejunal anastomosis was undone and normal continuity was established. Temporary inhibition of glycogenolysis of the liver may occur due to the transient high portal hyperglycemia following the rapid absorption from the intestine. Smith,[10] et. al., by doing insulin tolerance tests on patients found that patients liable to hypoglycemic attacks were characterized by an abnormal persistence of hypoglycemia on the intravenous insulin tolerance test. In our patients, 12 of 51 showed the late (hypoglycemic) symptoms and these were severe in only 4 instances.

AFFERENT LOOP SYNDROME

English authors refer to "bilious vomiting" as being separate from the usual early or late postcibal complaints. Wangensteen,[16] in 1945, and Ingelfinger,[17] in 1944, both pointed out that reflux of food into the afferent loop may be a cause of postcibal distress. The afferent loop syndrome is produced by gradual filling of the afferent loop with a mixture of food, bile, pancreatic secretions and intestinal secretions. With this filling of the afferent loop the patient will develop gradual postcibal distress which becomes more marked after each meal. After a few days he will vomit large amounts of liquid bile staining material not containing food and obtain relief for several days to weeks. The vomiting is often precipitated by changes in posture, especially stooping. The cyclic character of this symptoms complex is believed by some to be due to gradual accumulation of fluids in the afferent loop.

RECURRENCE OF ULCER

One of the most discouraging postgastrectomy findings is the recurrence of peptic ulcer. In our group of patients there were 5 patients who had a recurrent peptic ulcer following subtotal gastrectomy. Marginal ulcers most often occur on the jejunal side of the anastomosis and usually occur within the first 5 cm. of the jejunum or of the duodenum if the gastrectomy has been completed with a gastroduodenostomy. In some instances, however, the ulcer may be on the gastric side.

The cause of recurrences has been studied by many authors. Zollinger and Ellison [18] have recently demonstrated the existence of islet cell tumors of the pancreas containing Beta cells, occurring along with recurrent ulcers associated with high gastric resection. Since their original paper, other authors have also found that they had cases where recurrent ulcers following surgery have also been accompanied by islet cell tumors of the pancreas. These islet cell tumors have not been insulin producing tumors. Zollinger has postulated with inconclusive evidence that these islet cell tumors secrete glucagon and that the glucagon may be the ulcerogenic factor of pancreatic secretion. Zollinger and Ellison quote the work of Dragstedt [19] and Elman [20] in which these authors demonstrated that spontaneous duodenal ulceration nearly always occurs with the diversion of pancreatic secretions from the duodenum

while ulceration of the duodenum rarely occurs following total pancreatectomy. Poth [21] has shown that there is an increase in gastric secretion as the hyperglycemia induced by glucagon subsides.

The following two case histories from our group are typical examples of a severe ulcer problem with recurrence following gastrectomy. The first patient has not been investigated from the standpoint of pancreatic tumor and the second was investigated at the time of her surgery and no tumor could be found. At this time, however, it is the opinion of some of the surgeons who have seen her that she should have a subtotal pancreatectomy with the strong possibility that there is an islet cell tumor present.

I. C. 28 year old colored female, in 1953 had onset of ulcer type pain. Early in 1955 she had massive gastrointestinal hemorrhage and in March, 1955, a subtotal gastric resection with a gastrojejunostomy was performed. She had secondary surgery three weeks later because of an abscess in the abdomen. In December, of 1955, she had recurrence of pain with tarry stools and a diagnosis of a marginal ulcer. In January, 1956, she had a transthoracic vagotomy. In August, 1956, she was admitted to the hospital pregnant, with a severe anemia, and in September, 1956, delivered a normal infant. At this time she required numerous transfusions. In December, 1956, a 1.5 cm. ulcer crater was demonstrated distal to the stoma. She was placed on a medical regime but on December 29th, 1956, was re-admitted for the ninth time for a bleeding ulcer. On January 22nd, 1957, surgery was performed with a combined abdominal thoracic incision. The pancreas was completely explored and found to be normal. The gastric jejunostomy was resected and the gastric remnant anastomosed to the duodenum. On February 12th, 1957, she had abdominal surgery because of a suspected abscess and a white blood count of 49,000. No definitive procedure was done at this time. February 19th, 1957, she had massive bleeding again and at this time complete hematology studies revealed no abnormality other than hypercoaguability of the blood. On March 8th, 1957, she was feeling well and was discharged from the hospital. The surgeons suggested at the time of discharge that she should be re-admitted with consideration of a total gastrectomy and partial pancreatectomy.

J. C., 51 year old white male, who in 1947 gave a 6 year history of typical ulcer pain. In addition to severe ulcer pain he had become moderately addicted to narcotics. In 1947, he had a subtotal gastrectomy (Polya type). By July of that year he had recurrence of his ulcer pain and in October an ulcer was visualized at gastroscopy. By this time, he had a return of hydrochloric acid secretion with 1200 cc. of nocturnal secretion containing 100 units of free hydrochloric acid. In November, he visited another Clinic at which time an ulcer was not demonstrated and a bilateral splanchnic block was performed. He developed an abscess at the site of injection with no relief of his pain. In February, 1948, having continued to have pain, he had a massive hemorrhage and had to have several transfusions. Between February and June he was treated at his home with diet and again began to need narcotics. In June of 1948 he was treated with Enterogastrone without distinct benefit. He was given a rigid ulcer management without great relief and surgery was offered to him at this time. He declined. In October, he was admitted to a mental institution because of extreme anxiety and pain with uncontrollable emotional outbursts. At this sanatorium a mass was felt in his abdomen and the radiologist thought he had neoplasm in his stomach. In November, he went to Boston and had an initial operative procedure consisting of draining of the abscess and this was followed by a second resection of the stomach. His course since that time has been fairly good.

THERAPY

The treatment of patients with postgastrectomy symptoms is difficult and involves many factors. These patients frequently have a nutritional problem. Hayes [22] has been able to demonstrate that weight can be controlled if careful attention is given to diet. He points out that the oral administration of hypertonic glucose solution or protein hydrolysis produces an immediate increase in jejunal motility and these substances should be avoided. He also shows that in patients who have a gastric resection that the satiety value of high fat content in the diet it to a large extent lost and that high fat diets can easily be fed to these patients. He found that a diet of high fat, high protein content would be slowly hydrolyzed and should be fed with maximum exclusion of carbohydrates. The administration of liquids should be given between feedings. If the patient has discomfort soon after eating he may be relieved by lying down after meals. Some patients may even have to eat their meals lying down.

Mills [23] has shown that due to mechani-

cal reconstruction following gastrectomy, bile and pancreatic juices must pass through the stump of the stomach before entering the efferent loop. This volume of food is augmented by the gastric secretion from the stump of the stomach and saliva. A conservative estimate of the combined secretions passing through the stomach would amount to 2 to 3,000 cc. A high percentage of this fluid is secreted at meal times during the day. The volume of these juices plus food and liquid ingested is more than the stump can accommodate. He feels that a dry diet is for this reason quite important, and he recommends a dry diet with 4 to 5 feedings per day. He finds that a carminative mixture given one-half hour before meals tends to trigger the gallbladder and stimulate the pancreatic juices and then these secretions go through the stomach ahead of the meal in a more normal physiological relationship. Some of his patients used beer for this purpose.

Various drugs have been used to treat the early postgastrectomy complaints including the anticholinergic to slow down motility. Potassium chloride is used with the idea that benefit may be obtained by preventing the lowering of potassium levels in the blood that have been postulated. The ingestion of large amounts of liquids, one-half hour to twenty minutes before meals, may prevent the decrease in blood volumes that occur in some instances. Urecholine has been used with some benefit perhaps because of its effect on emptying the afferent loop. Glucagon has been used with some benefit in relation to the early complaints, probably because of its effect on reducing gastric motility. (Thomas Witten, Denver VA,[24] personal communication.)

The treatment of the late postprandial complaints, namely the hypoglycemic symptoms, is more difficult. Frequent feedings will help to prevent the onset of hypoglycemia and rest is often helpful. When this complaint is severe it becomes quite disabling since activity increases the frequency and severity of these symptoms. Fortunately, this abnormal physiological situation usually disappears within the first year following surgery.

The treatment of the anterior loop syndrome consists of measures to empty the afferent loop and if possible to prevent the accumulation of material in this blind pouch. Lying down one-half hour prior to meals and the use of carminative mixtures before meals may help to prevent the development of progressive nausea. The use of dry feedings is frequently beneficial. Capper and Welbourn[25] recommend sodium bicarbonate taken one-half hour before meals to stimulate the emptying of the duodenum.

If the symptoms of the postgastrectomy syndrome persist and are disabling surgical intervention may be indicated. The surgical procedures which are most frequently employed are conversion of gastrojejunostomy to gastroduodenostomy, revision of the stoma, and supporting the lesser curvature.

SELECTION OF PATIENTS FOR SURGERY

A most important factor in prevention of severely disabling postoperative complaints is the selection of proper patients for surgery. Patients who have been seriously ill or have serious complications from ulcer will usually tolerate postgastrectomy complaints well. Patients who are operated upon for minimal indications will tolerate postgastrectomy complaints poorly. It is difficult to generalize but it is probably safe to say that the younger patient with a high nocturnal secretion is more likely to have a recurrent ulcer than the older patient with the low acid value. The nervous, high strung individual with a labile vascular system is more likely to develop postgastrectomy complaints of a more severe nature than the placid individual. The well-nourished person is less likely to have serious nutritional problems postoperatively. Surgeons usually state that the surgery is a failure when the postoperative complaints are more severe than the original ulcer. With the knowledge that the removal of part of the stomach may produce symptoms, the pa-

tient with an ulcer should not be treated surgically unless his symptoms are severe and intractable or threaten his life.

The problem of intractability of the ulcer or the patient presents a serious problem. The intractability of the ulcer should in most instances be the determining factor. An intractable patient without serious ulcer complications is likely to be very difficult to manage if he has postgastrectomy complaints. The following case history presents an illustration of intractability.

White man, 42 years old, was admitted to the hospital with an ulcer type pain, relieved by food and alkali. He had a previous diagnosis, two years before, of a duodenal ulcer and had one episode of mild hemorrhage. In the succeeding two years he had had recurrences of his ulcer during episodes of heavy drinking. For six weeks prior to his admission to the hospital the intake of food had been inadequate and his intake of alcoholic liquor excessive. In addition, he had several encounters with the law. At the time of admission to the hospital he had been transferred from the jail to the hospital. Because of the above history the surgeons felt that this was an intractable ulcer and a subtotal gastrectomy was performed. The patient had a relatively uneventful postoperative course and was discharged back to jail and served a two year federal prison sentence. During the time of his imprisonment he had few gastrointestinal complaints. On his release from prison he continued his previous extracurricular activities including heavy drinking, and excessive smoking. He ate poorly and had numerous exciting encounters with the police. In this situation he developed recurrent abdominal pain, nausea, vomiting, and had two massive gastrointestinal bleeding episodes. On return to the hospital he gradually responded to medical management and after several months was in condition to be discharged. At this time he was thirty pounds under his average weight. He was not subject to further imprisonment and without the security of prison he had a poor prognosis.

This patient illustrates the fact that if the intractable personality can be controlled the patient will respond well to medical or surgical therapy. Medical treatment of ulcer in an intractable patient does not have the potential risk that surgery has in the intractable patient with an uncomplicated ulcer.

Careful selection of patients for surgery is the best means of avoiding serious postgastrectomy problems. Recommendations for surgery should be based on the pathology not the personality. The intractable person may develop such serious complications of his ulcer that removal of part of the stomach is necessary. Occasionally this type of patient will have no postoperative problems. The postoperative patient will be happy despite some discomfort if his original disease was severe.

SUMMARY

A discussion of the theoretical factors in relation to the postgastrectomy symptoms has been presented along with a summary of the findings in 51 patients with postgastrectomy complaints. To date there is no uniformly successful treatment of the postgastrectomy symptoms. Careful evaluation of the patient's symptoms and detailed attention to their diet and medical management will, however, bring about a satisfactory result in the management of most patients. Great care in selection of patients for surgery is the best prophylactic measure in preventing disabling postgastrectomy results.

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TRAUMATIC VENTRICULAR SEPTAL DEFECT: REPORT OF A CASE TREATED SUCCESSFULLY *

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Defects of the ventricular system occur as a relatively common congenital malformation.[1] They were formerly of little surgical significance, inasmuch as a reasonably safe approach to their closure had not been developed. In recent months, however, with the use of several types of extracorporeal circuits, it has been possible to effect a closure of such defects within a reasonable range of mortality. Acquired ventricular defects by nature of their etiology carry a far more serious prognosis. They may result from dissolution of a portion of the septum secondary to the infarction of coronary thrombosis,[2] or they might result from trauma, usually from a penetrating type wound, although rupture of the septum might occur with a crushing or nonpenetrating type of injury. Bender[3] recently reported a case of an acquired ventricular septal defect which was secondary to blunt trauma of the chest, but which was not evidenced until two weeks following the accident, and was thought to be a late perforation of the septum secondary to ischemic necrosis. Defects of the septum secondary to myocardial infarction are of course extremely poorly tolerated and to our knowledge, successful closure of such a defect has not been achieved, the life expectancy following their appearance usually being measured in days rather than months or years. We have not observed a defect due to nonpenetrating trauma. Recently, however, a case was admitted to the Tulane Surgical Service of Charity Hospital in which a clinical diagnosis of traumatic ventricular septal defect was made, and in which successful closure of the defect was achieved utilizing techniques similar to those employed in correction of congenital defects. The case will be reported in detail.

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CASE REPORT

M. D., a 21 year old Negro male was brought to Charity Hospital on July 1, 1956, approximately fifteen minutes after having been stabbed in the left upper chest anteriorly. No blood pressure was obtainable on admission although a weakly perceptible peripheral pulse was noted and the heart sounds could be ausculated. Breath sounds were markedly decreased over the left chest and there was dullness to percussion in the same area. Films of the chest confirmed the presence of a large hemothorax on the left. A catheter was inserted into the left chest and attached to water-seal drainage and approximately 2300 cc. of blood recovered. Because of the swelling about the site of the stab wound (second intercostal space just outside the midclavicular line) and suggestive diminution of the left radial pulse, the possibility of laceration of the intrathoracic left subclavian artery was entertained, and the patient was immediately taken to the operating room having received 2000 cc. of whole blood. Following this blood replacement his blood pressure had risen to 140 mm. Hq. systolic.

Operation: The patient was anesthetized with cyclopropane, and upon insertion of an endotracheal tube, cardiac arrest occurred. Without sterile precautions the left chest was immediately opened through the third intercostal space, transecting the sternum, and with division of the left third and fourth costal cartilages. The heart was dilated and in asystole but spontaneous rhythm returned after "massaging" the heart three times. The left pleural space was noted to be filled with blood and there was active bleeding from a perforating wound of the left upper lobe. In addition blood was noted to squirt from a 1.5 cm. laceration in the pericardium with each contraction of the heart. While controlling the bleeding from the left upper lobe with pressure, the pericardium was widely incised and a laceration approximately 1 cm. in length in the outflow tract of the right ventricle was observed. A moderate hemopericardium was present. *The blood emitting*

from the ventricle was noted to be dark in color. The laceration in the heart was closed with interrupted 000 silk Lembert sutures which controlled the bleeding. The wounds in the left upper lobe were sutured similarly. At this point, the patient, having received 17 pints of whole blood, manifested acute pulmonary edema. Edema fluid was aspirated from the endotracheal tube and the heart was somewhat dilated. The patient was digitalized with cedilanid in divided doses; positive pressure inflation of the lungs was continued, and approxmately 400 cc. of blood was withdrawn from the venous circulation with noticeable improvement. The pericardial sac was loosely approximated, bleeding from the chest wall controlled, and two indwelling chest catheters brought out through stab wounds for drainage of the pleural space.

Postoperatively, the patient did fairly well. He had a moderate elevation of temperature the first week, and ran a low grade temperature for approximately fourteen days thereafter. He exhibited a moderate tachycardia for the first two weeks postoperatively. He began taking a diet promptly and his ambulation was progressively increased.

Serial ECG's were made. Initially a sinus tachycardia was the only finding. Later changes suggestive of pericarditis were observed, and still later a delay in right bundle branch conduction was noted. As early as the second postoperative day a rather harsh systolic murmur was audible over the pericordium, and subsequently, a systolic thrill could be felt in the same region. The possibility of this representing the murmur of an acquired ventricular septal defect was entertained. However, the patient continued to improve following surgery and showed no further evidence of congestive failure. He was discharged from the hospital four weeks after admission.

The patient was readmitted to the hospital approximately two weeks later for further diagnostic studies, chiefly cardiac catheterization. Review of the patient's old records revealed hospital admissions at ages 9 and 19, at which time no murmurs had been heard and the heart sounds were normal.

Cardiac catheterization* was performed on August, 24, 1956, (Fig. 1) approximately eight weeks following the stabbing, and revealed evidence of a left to right shunt at the ventricular level, localized in the outflow tract of the right ventricle just below the pulmonary valve. The pulmonary blood flow was calculated to be 10 liters/min., as compared to a systemic flow of 6.6 liters/min. Moderate elevation of the pulmonary artery pressure was observed, (36.2/18) but the pulmonary arteriolar resistance was normal (calculated as 80 dyne cm.[5]). Although the measured shunt was small

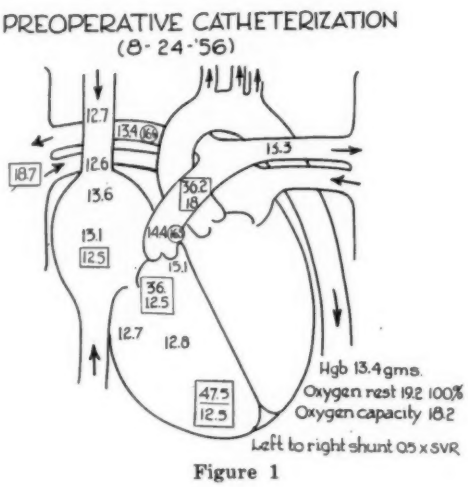

Figure 1

and the patient had no symptoms, it was felt that over a period of time a further increase in the pulmonary artery pressure would occur and surgical closure of the defect was indicated.

Operation: On August 31, 1956, operation was performed (O.C.) utilizing endotracheal anesthesia (N_2O) plus sodium pentothal(R) and anectine(R) intravenously. A bilateral anterior thoracotomy was made in the fourth intercostal space, transecting the sternum. The pericardium and great vessels were exposed and the pericardium opened. There was a very extensive inflammatory process present, almost completely obliterating the pericardial space, and rendering dissection quite difficult. The region of the previous wound in the right ventricle was exposed and a marked thrill was palpable over this area. The superior and inferior vena cavae were dissected free and tapes placed about them to achieve subsequent inflow occlusion. Following the administration of 110 mg. of heparin, a Cournand needle was inserted percutaneously into the left femoral artery in order to monitor the blood pressure. The right femoral artery was exposed through a small incision in the groin and a polyethyelene catheter (3/16" I.D.) was inserted proximally and held in place by occluding tapes to serve as an inflow route of arterialized blood. Similar catheters were introduced into the superior and inferior vena cavae via the right auricular appendage and so placed that the cavae could be occluded proximal to their openings. The inflow and outflow catheters were connected to the respective ends of a bubble-oxygenator patterned after that of DeWall.[4] The extracorporeal circuit was established and the cavae were occluded. The right ventricle was opened along its outflow tract and held open by traction sutures.

A defect measuring approximately 1 cm. in diameter and located about 2 cm. proximal to the

* Cardiac catheterizations, preoperatively and postoperatively, were done by Dr. Albert Hyman.

pulmonary valve was found. This was closed with five interrupted sutures of 000 silk. The incision in the ventricle was then closed with a continuous 000 silk suture permitting the air in the chamber to escape through the incision. A second suture was used to effect satisfactory hemostasis. The caval tapes were released allowing venous inflow to the heart to be resumed and the pump was gradually slowed as the cardiac action and peripheral pulse and pressure improved. The ventricle was open for a period of six and one half minutes and the perfusion lasted for sixteen minutes. The cavae catheters were removed and the auricular appendage ligated. The pericardium was loosely approximated and a tube was left in each pleural space for drainage.

An infusion of protamine sulphate was started by slow intravenous drip and discontinued after forty-five minutes when the clotting time was measured as five minutes. The catheter in the right femoral artery was removed and the artery was repaired with a continuous 00000 silk suture. The chest wall was closed in the usual fashion, utilizing interrupted wire sutures to approximate the sternum.

At the conclusion of the procedure the patient's general condition was satisfactory although he manifested a tachycardia in the range of 150. A tracheotomy was performed primarily as a precautionary measure and the patient was transferred to the recovery suite. A total of 2500 cc's blood loss occurred, chiefly from the right ventricle (coronary sinus).

Hospital Course: The patient recovered consciousness promptly. Except for a moderate tachycardia (130-150/min.) and slight tachypnea he manifested no outward effects of the procedure. The murmur in the precordial area could no longer be heard. Subsequently, the patient exhibited a moderate febrile response along with his tachycardia, and had some difficulty with bronchial secretions. However, the tracheotomy tube could be removed by the fourth postoperative day. On the eighth postoperative day approximately 60 cc. of fluid was aspirated from the right chest and a localized collection of seropurulent material evacuated from his wound. Subsequently his temperature fell to approximately normal levels and his tachycardia was less marked. Cultures of the wound and pleural fluid failed to grow any organisms. The patient's course was one of general improvement thereafter, and he was discharged from the hospital approximately twelve days later.

At the time of his discharge his vital signs had been normal for several days and he was completely asymptomatic. Cardiac catheterization was repeated on November 20, 1956 (Fig. 2). No evidence of a shunt, either left to right, or right to left could be demonstrated at this time. The pressure in the pulmonary artery remained somewhat higher than normal as did the pressure in the right ventricle. However, it is felt that this is a

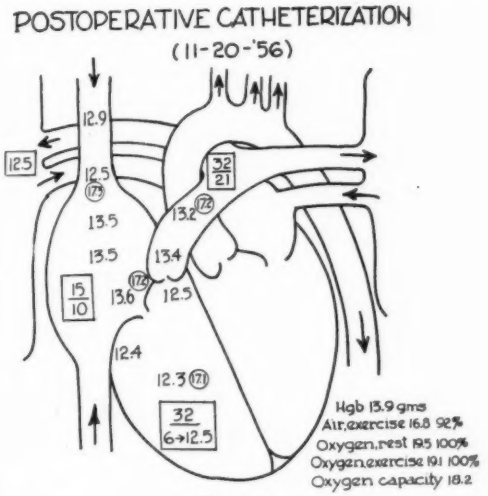

Figure 2

temporary elevation and will return to normal levels over a period of months.

DISCUSSION

To our knowledge there have been only two other patients in whom a diagnosis of traumatic ventricular septal defect was made clinically and in whom an attempt to close the defect was undertaken. Campbell [5] reported a successful closure of a traumatic ventricular defect utilizing a heterologous lung oxygenator; whereas Cooley [6] was unsuccessful in an attempt at closure by a modification of the Gordan-Murray technique. With respect to surgical closure of a ventricular septal defect secondary to myocardial infarction, we know of but a single instance in which such was attempted but the closure was apparently unsuccessful; the patient died in the early postoperative period.[7] Examination of the heart at autopsy revealed that the Ivalon patch had become detached.

It is interesting to conjecture as to whether or not the defect in our patient might have closed spontaneously. At the time of operation, two months following the original injury, there was no evidence that the defect was healing. Its appearance was somewhat ovoid as opposed to the probable slit-like character at time of injury, suggesting secondary changes in the hemodynamics of the right ventricle

might be tending to favor perpetuation of the lesion, namely, the increased right ventricular pressure and/or dilatation of the right ventricle. Kay produced defects of the ventricular septum in dogs using a cork-borer technique. He found that defects measuring 3/8" in diameter healed spontaneously in two to five months in approximately 20 per cent of the long term survivors.[8] Griffin and Essex[9] found that all four animals with 8 mm. defects produced by a "punch" showed spontaneous healing within four months' time. Of 5 animals with 10 mm. defects, they found 3 healed at the end of one month, 1 healed at the end of two months, and described the last as nonfunctioning at the end of one month. Two animals in which defects 12 mm. in size were produced died within eight days.

The incidence of traumatic defects of the ventricular septum is quite low, and the mortality is undoubtedly increased when this factor is superimposed on a penetrating or perforating wound of the heart. Biggers[10] reported 17 cases of heart wounds; none with involvement of the septum. Elkin[11] reported 23 cases with no septal wounds evidenced. Valle[12] reported 19 cases of foreign bodies retained within the heart, three of which were thought to lie within the septum and none of which was successfully removed. Maguire and Griswold[13] reported 33 cases of penetrating wounds of the heart and pericardium, four of which had injuries to the ventricular septum. All four of these cases died. Maynard,[14] in 1952, reviewed 81 cases of penetrating wounds of the heart and described no cases in which a diagnosis of septal perforation was made. In 1956, Maynard reported on 43 additional cases of heart wounds, and described one case in which an injury of the atrioventricular septum was found at autopsy. Priario[16] reported a case of a gun shot wound of the heart in which the missile apparently traversed the interventricular septum without producing a communication between the ventricular cavities. It was necessary to ligate the posterior descending coronary artery. No murmurs were detected postoperatively, and serial electrocardiograms were interpreted as normal.

Perforation of the infarcted interventricular septum occurs with far greater frequency than the traumatic injuries, there being over 70 cases in the literature in 1951,[2] and the diagnosis was made antemortem several times. However, the lack of myocardial reserve and the underlying disease of the coronary arteries make the over-all prognosis in this group much graver than in the traumatic group. Nevertheless, the extremely poor prognosis with this type of lesion warrants continued efforts to salvage some of the patients.

SUMMARY

A case is reported in which a 21 year old colored male sustained a penetrating wound of the heart, as well as of the interventricular septum. The perforation of the septum was manifested in the early postoperative period by a characteristic murmur and thrill and was confirmed by cardiac catheterization. Approximately eight weeks following the original injury successful closure of the defect was achieved. Subsequent cardiac catheterization no longer demonstrated a shunt existing between the ventricles. Acquired defects of the ventricular septum are briefly discussed.

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SURGICAL TREATMENT IN CEREBRAL PALSY *

DANIEL M. KINGSLEY, M. D.
ALEXANDRIA

Surgical treatment of cerebral palsy is not a cure-all. Indeed, surgery plays a relatively minor role in the over-all plan of rehabilitation of this condition. Nevertheless, in that small group of patients amenable to surgical help, operative procedures are important.

In the upper extremities, surgery may make the difference between ability or inability to dress, feed, and obtain employment. In the lower extremities, surgery may enable a patient who has never walked and who has been confined to a wheel chair, to get up and ambulate under his own power.

Certain surgical procedures, such as arthrodeses of the wrist or feet, heel cord lengthenings, adductor tenotomies, and neurectomies at various levels have been standard practice for many years. However, authorities in this field have discouraged any wide-spread endeavors to extend the indications to other procedures. Only in the past four or five years has there been a generalized renaissance of orthopedic surgical procedures in cerebral palsy. Newcomers to this field have cautiously proceeded to extend the operative indications. The successful results have advanced the number of indications for surgery to a marked extent. Even athetoids, in whom surgery has supposedly been contraindicated, have repeatedly been greatly benefited.

Personal experiences at the State School for Spastic Children over the past eight years has afforded an opportunity to perform a variety of procedures sufficient to lead to some tentative conclusions. Certain of these will be presented in this discussion.

UPPER EXTREMITIES

In the upper extremity, only a few patients have been operated upon. One procedure which promises to be useful, is correction of pronation contractures of the forearm. This type of contracture is frequently seen in hemiplegics. At surgery the pronator teres is found to be tightly contracted. It jumps like a steel spring when released. Such release is not sufficient in itself. A motor must be placed in position for supination of the forearm. Two steps have proven helpful. The more important one is transfer of a useful, functioning muscle, usually the flexor carpi ulnaris, to the dorsal and radial aspect of the distal portion of the radius. In addition, the pronator teres is attached to a cuff of periosteum carried around from the anterior surface of the radius to the radial side of the radius, and then dorsally, to project as a tongue on the dorso-ulnar aspect of the radius. The pronator teres is thus salvaged for use as a motor for supination.

LOWER EXTREMITIES

In the lower extremities, in addition to the operations already mentioned, several procedures have repeatedly proven useful. It is necessary to start rehabilitation at the hips and proceed distally, to the knees and ankles. This plan is necessitated by the postural reflexes set in motion by hip deformities, such as flexion contractures. These produce knee and ankle flexion and consequent gait disturbances. Any deviations from this plan lead to complications.

*Presented at the Seventy-seventh Annual Meeting of the Louisiana State Medical Society, May 8, 1957, New Orleans.

In two limbs, correction of flexion contractures of the hips and knees was done first at the knees, as advocated by Eggers. The knees were successfully straightened, but the hips were not improved. It was impossible to use braces past the thighs for standing or walking in these two limbs. These results discouraged any changes in the overall plan.

Similarly, correction of hip contractures alone proves contraindicated when knee flexion contractures are also present. The latter must be corrected before braces can be applied to hold correction of the hips.

Hip and knee flexion contractures frequently are found associated. The hips are corrected surgically, after physical therapy has gone as far as possible and proved insufficient. Smith-Petersen incisions are usually utilized to locate tight structures, such as tendons, ligaments, and hip joint capsule. A Luck incision was used twice, with good results. A double hip spica cast is used for six to eight weeks depending on the clinical degree of spasticity. At three weeks the patients have usually recovered enough to allow correction of knee contractures when the latter are indicated also. The double hip, long leg spica cast is then re-applied. When the knees alone are operated upon, a long leg cast suffices, with the knees held in complete extension for six to eight weeks.

Long leg braces with pelvic bands and hip and knee extension locks, are then used for prolonged periods, together with physical and occupational therapy. At night, plaster beds are frequently utilized to afford rest to pressure points resulting from the braces. Gravity positioning on stretchers during school hours, has proven useful.

KNEE FLEXION DEFORMITY

In the knees a flexed position may be due to one of three causes: (1) quadriceps weakness; (2) flexion contracture of the hamstrings; (3) the result of balancing reflexes to accommodate to a hip flexion contracture. Flexion contractures due to the hamstrings are the most common type. At first, correction by Eggers procedures was done. For the past two years, however, equally satisfactory results have followed adequate detachment of all hamstrings without any special attempts to cut the retinacula or to reimplant the cut tendons into the femur.

One theoretical danger attached to this release of all the hamstrings, is resulting overaction of the quadriceps. This effect has not occurred. However, occasionally a patient who has not been subjected to any surgery whatsoever, is seen with patellae pulled far cephalad. The mechanism for producing this result is unknown, but obviously the patellar tendons must have yielded to a continual stretch during the growth period. In these patients, quadriceps weakness is present and adds to the confusion, since a strong quadriceps might otherwise be considered as the cause of the patellar displacement.

EQUINUS GAIT

Hip flexion contractures are especially pernicious in causing subsequent knee flexion and then one of the three types of equinus gait found in cerebral palsy follows. The key to this commonly observed condition is the angle between the leg and foot, at the ankle. This is actually in a calcaneus position in such patients. Yet this type of patient cannot walk, except on his toes, with the heels failing to touch the floor. When these patients are braced solidly, with equinus stops added, they lose their previous ability to walk. In such patients, the braces are frequently therefore made still stronger. It is then noted that the shoes become deformed. The heels do not contact the bottom of the shoes and ambulation is still impossible. Pressure sores often result from these braces, before they are discarded as useless. In these patients, the equinus gait is automatically corrected when the hips and knees are extended by surgical methods already described. Tragic results follow lengthening of the heel cords in an attempt to correct this equinus gait. The calcaneus already present, but concealed, is thereby increased to such an extent that

walking becomes totally impossible.

Another, similar, type of equinus gait is due to quadriceps weakness. Bracing relieves this abnormality of gait. Sometimes the weakness is so slight that the time-tested method of strengthening the quadriceps muscle by a heel raise alone, is sufficient for correction.

There is a reciprocal interdependence between the knee-flexion position and the calcaneus of the feet. Both are related as a response to gravity, through the semicircular canals. When the knees are held flexed, it is extremely difficult to ambulate except with the foot in calcaneus. Yet the heels must be elevated to give a push-off. The weight is then borne on the toes and forefeet alone. When this combination is seen, of calcaneus at the ankle, yet of weight being borne on the forefoot with the heel held off the floor, the conclusion is inescapable that there must be knee flexion causing the observed gait. Further analysis as to the presence of a knee or hip flexion contracture, or of quadriceps weakness alone, then leads to proper treatment by physical therapy, bracing, and surgery.

A second type of equinus gait in cerebral palsy is a result of a truly shortened heel cord. This is easily determined by tests in common use. This type is about as frequently encountered as the previous one. Associated with this second type of equinus is a deformity of gait, with eversion of the heels, but the heels contact the floor. This causes various extremes of flatfoot, all characterized by heel eversion, sag of the arches, and rapid deformation of the shoes. The cause of this equinus is frequently concealed when the heels bear weight in eversion. Advanced degrees of equinus without eversion of the heels and flatfoot, and with weight borne only on the forefoot, have not been encountered.

This type of equinus gait must be differentiated from one which is identical except for etiology, since there is no equinus actually present in the patients under discussion. Instead, there is overaction of the peroneal muscles. Eversion of the heels, sag of the arches, and foot deformity are similar to the concealed equinus gait just described. In both, the heels bear weight.

Treatment is dependent on proper analysis. In the hidden type of equinus, heel cord lengthening corrects the deformity. In types which do not require much lengthening, the gastrocnemius can be loosened at its popliteal origin and slid distally by a recession type of operation. In patients who need more length, a Hibbs type of heel cord lengthening is done, sometimes combined with posterior capsulotomy of the ankle joint.

On the other hand, the flatfoot due to peroneal over-action is corrected by the transfer of the peroneus longus to the position of the anterior tibial muscle.

The third, and last, type of equinus is seen less commonly than the other two types. It is the result of over-action by the posterior tibial muscles. So far, physical therapy has been sufficient for correction, without resort to surgery.

PRACTICAL VALUE OF MEASURES

The application of the principles enunciated here has been of increasingly greater practical value as time goes on. It is encouraging to be able to examine the static and dynamic posture, then do a muscle check, and from these findings, outline a program of physical therapy, bracing, and surgery that leads to self-sufficiency. The considerations mentioned here are rewarding especially from the standpoint of proper bracing. Braces for these patients are among the most expensive devised. It is therefore gratifying to be able to prescribe at the outset, braces that help, instead of hinder rehabilitation. The proper analysis of physical therapy to be used is another saving of energy and leads to continued enthusiasm on the part of both patients and personnel. Finally, tragic sequelae of technically good, but improperly indicated, operations are avoided.

In this series of patients, neurectomies have not been performed. It has always

seemed more logical to utilize the muscle pulls by transferring them rather than to weaken them irretrievably by sectioning their nerve supply. Perhaps the final proof of this opinion is the fact that patients with extensive neurectomies still have the deformities described in this paper. The procedures advocated here had to be performed before these patients could be rehabilitated.

Secondary rewards of more rapid mental progress and improved family and social relationships, frequently overshadow the primary surgical benefits. Improvement to an extent allowing employment in industry occasionally results, but is not to be expected. Nevertheless, decrease in physical and financial burdens on the part of families or institutions is rewarding.

CONCLUSIONS

After all other modalities of treatment have been tried to rehabilitate patients with cerebral palsy, surgery is found to salvage a relatively small percentage, in whom other methods have failed.

Surgery is indicated especially in fixed deformities that do not yield to physical therapy. Such deformities are frequently found. Most of the standard methods of correction are omitted because they are so well known. The procedures which are less often used, but which have stood the test of time in producing rewarding results, are described.

In the upper extremity, muscle releases and transfers are done for pronation contractures of the forearm.

In the lower extremity, rehabilitation starts at the hip and proceeds distally. The inter-relationship of hip and knee flexion contractures with posture of the ankles and feet, is stressed and analyzed.

Three types of knee flexion deformity are discussed.

Three types of equinus deformity of the feet are analyzed.

Two types of flatfoot are described.

Treatment is outlined for each type.

Stimulation of the mind after surgical rehabilitation often leads to startling mental progress, with improved family and social relationships. Nevertheless, improvement to a degree compatible with self-sufficiency outside of a sheltered environment, is rarely attained.

CORONARY ATHEROSCLEROSIS *

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Coronary artery disease is too broad a subject to attempt to cover in one hour and I have decided to limit this discussion to atherosclerotic disease of the coronary arteries. I have done no research on the etiology of this disease and plan none in the immediate future. Having thus qualified myself, most of my remarks may be regarded as in the light of an unbiased opinion.

FACTORS IN INCREASED INCIDENCE

There can be little doubt that an actual increase in the incidence of atherosclerotic coronary disease has gradually developed during the past thirty years. Several factors appear to have contributed to this increase. In this same period, longevity has increased by about ten years. Thus, larger segments of a larger population have entered the age period in which atherosclerosis in general, and of the coronary arteries in particular, is known to be more common. There has developed a considerable increase in our general knowledge of coronary disease in this same time period and the extensive use of the direct writing electrocardiograph, particularly since the second World War, are factors which greatly contribute to much more accurate diagnosis in this area. Pathologists are not unlike clinicians in that they can also find "what they look for." Moreover, the greater segment of the population in the advanced age period over sixty years has required considerable therapy in the field of major surgical procedure for disease or trauma unrelated to

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* Transcription of a discussion given at the Shreveport meeting of the Louisiana State Heart Association, May 15, 1957.

† Professor of Medicine, University of Oklahoma School of Medicine.

the cardiovascular system, and the associated incidence of shock has served to precipitate myocardial infarction in many a patient who might have otherwise avoided the clinical level of coronary artery disease. Finally, I am informed on good authority [1] that the fat content of the American diet has probably doubled in the past thirty years and there is reason to believe this may be a contributing factor to the increased incidence of coronary atherosclerosis.

ALTERED BLOOD LIPID PICTURE

For the past seven years, there has developed a growing conviction amongst cardiologists that clinical atherosclerosis is not a necessary consequence of age. A view of this kind is certainly important for both the researchers and practicing physicians who together look for cause with a view to prevention or specific treatment. There has now accumulated considerable data which indicates that atherosclerosis may be associated with an altered blood lipid picture. A brief review follows. In 1953, Barr [2] pointed out that, among all the mammals, only man was subject to atherosclerosis and only in man was one confronted with relative high values of certain factors of the blood lipid picture. These factors are the total serum cholesterol, the cholesterol esters, the cholesterol - phospholipid ratio, increased concentration of the B_1 lipoprotein aggregates, and increased concentrations of the lipoprotein macro - molecules of the S_f 12-20 and 35-100 class.*

In 1950, Gofman [3] pointed out that only infants were immune to atherosclerosis; that children and young adults and women in the childbearing age period were relatively immune and that in the immune and the relative immune groups there are relatively low concentrations of the "specific" classes of lipoproteins. On the contrary, he found increasing concentrations of these aggregates in man through the sixth decade, particularly in subjects having spontaneous atherosclerosis. In 1953, Barr [2] showed that estrogens could reverse the altered blood lipid picture and two years later Russ [4] demonstrated that androgens produced an opposite effect. Also, in 1953, Wuest, Dry and Edwards [5] showed that there was an increased incidence of atherosclerosis in women following surgical menopause.

There are a number of diseases which tend to show the altered blood lipid pattern in one form or another and which are known to be associated with severe atherosclerosis. These diseases are diabetes mellitus, familial cholesterolemia or familial hyperlipemia, myxedema, nephrosis and xanthomatoses.

On the experimental side of the picture, a considerable number of investigators have been able to produce experimental atherosclerosis in a variety of animals. This has uniformly been done by feeding high cholesterol diets often, in addition to other specialized procedures. In the omnivorous animals, the distribution of experimental atherosclerosis is the same as it is in man. Only recently, however, experimental atherosclerosis has been followed by actual myocardial infarction.[6] In 1910, Windaus,[7] and again in 1933, Cowdry [8] demonstrated that most of the deposition of the early atherosclerotic lesion was composed of cholesterol.

HIGH FAT DIET

On the dietary side of the picture, evidence continues to accumulate. In 1946, Steiner [9] pointed out that there was a low incidence of diseases attributable to atherosclerosis in vegetarian groups, particularly those on a low fat diet. In 1947, Wilens [10] showed that severe atherosclerosis was ten times more common in the obese than in subjects of normal weight at comparable age periods, and was twice as common in hypertensive as in normotensive subjects. In 1952, Keys [11] emphasized the importance of the total fat content of the diet rather than that of the cholesterol per se. He was much impressed by the low incidence of atherosclerosis in the Bantu of South Africa

* These figures are flotation rates in centimeters per second per unit field of force in the ultracentrifuge.

who subsist on a diet in which only about 10 percent of the total calories is contributed to by dietary fat.[12] In 1952, Gofman and Jones[13] showed that there was an increased concentration of lipoproteins of S 35-100 class in obese subjects as compared with that in lean subjects. Gofman, et al[14, 15] also showed that prolonged low fat diet gradually reduces the concentration of abnormal lipoproteins.

During World War II it was not possible to obtain a rich diet high in fat and during this period there was a sharp decline in diseases attributable to atherosclerosis.[16] In comparison with other regions of the world there is a high incidence of diseases due to atherosclerosis in the dairy farming countries, the United States, Great Britain and Denmark.[17]

In 1951, Wilens[18] showed that under given filtration pressures almost all of the inorganic constituents of serum could be made to filter into the arterial walls from the lumen outward. Cholesterol was present in the filtrate in low concentration and limited to the subintimal region of the arterial wall, stopped as it were, by the internal elastic lamina. Undoubtedly, observations of this kind led Page[19] (1954) to elaborate his filtration theory of atherogenesis. The implication is that normal subjects gradually deposit a limited amount of cholesterol in the subintimal region, but when there occurs an increased concentration of those lipid particles best adapted to penetrating the arterial intima, the rate and quantity of deposition is greatly increased, particularly if the filtration pressure (blood pressure) is high or if the intima has increased its permeability due to a change in its ground substance. In 1952, Moon and Rinehart[20] showed that most atherosclerotic lesions were preceded by a local alteration in the ground substance of the intima which is followed by a fibroblastic hyperplasia. In 1953, Kats and Stamler[21] felt that the thyroid hormones and potassium iodide offered some protection to experimental atherosclerosis, possibly because of a decreased permeability of the vascular intima which is attributed to the action of these drugs.

PREVENTIVE MEASURES

It should be remembered that the clinical evidence is largely statistical with great overlapping on either side of the argument. Nevertheless, it is natural that evidence of the kind indicated should be followed by suggestions regarding the possible prevention or specific treatment of atherosclerosis at the clinical level. These suggestions are: use of one or more of the following measures, the low calorie diet, the low fat and/or cholesterol diet, the lipotrophic agents, the plant sterols, heparin and heparin-like substances, estrogen, and finally thyroid hormones and potassium iodide.

Certainly, the low calorie diet is indicated for the treatment of obesity whether or not there is clinical evidence of atherosclerosis. The life insurance companies have been long aware of the danger of obesity. There is, I believe, a twenty-year less life expectancy for those who enter the forty-year period fifty pounds or more over ideal weight. It appears advisable to slowly reduce obese subjects with coronary atherosclerosis by low calorie diet. To reduce them rapidly is to put them on a high-fat diet, their own body fat. Some cardiologists[17] have been impressed with the occurrence of acute myocardial infarction in subjects who have recently lost considerable weight in a short period of time.

The low-fat diet appears to be a reasonable therapeutic procedure in treatment of coronary atherosclerosis. For this purpose it is suggested that no more than 20 to 25 per cent of the total calories be contributed to by dietary fat. At the present time it is not known whether the fat content should be animal fat or vegetable fat, or both.

The lipotrophic agents choline, methionine and inositol are extensively used. These substances have been known to be useful in the treatment of fatty liver due to certain deficiency states. It was hoped their use might reduce the cholesterol-phospholipid ratio. Clinical evidence is in-

sufficient and contradictory and in general the lipotrophic agents are not recommended for therapy in coronary atherosclerosis. As a matter of fact, choline is now on the atherogenic list.[6]

Plant sterols as yet have not proved to be of definite value. Consequently, beta sitosterol, dihydrocholesterol, cholesteryl chloride, other cholesterol analogues and special vegetable fats are not recommended as specific therapy. Moreover, the clinical evidence is equally weak for the use of heparin or heparin-like substances. The experimental evidence is confined, I believe, to work with the rabbit.

Estrogens in experimental and clinical use for atherosclerosis have been uniformly confined to the synthetic preparations rather than the natural hormone.[22] Their use in man produces irritability, depression, femininity, and impotency and there is as yet no evidence that the course of coronary atherosclerosis in man can be altered. These substances are consequently not recommended.

Clinical evidence to support the use of the thyroid hormones and/or potassium iodide is totally lacking and these drugs are therefore not recommended for specific therapy in coronary atherosclerosis. The present phase of our discussion terminates with the argument that, as yet, there is no *specific* therapy for coronary atherosclerosis.

CLINICAL PHASES

Let us turn our attention now to the clinical phases of the problem. If we examine a sufficiently large number of human hearts, all of which show severe atherosclerotic occlusive changes in the larger segments of the coronary arterial tree, there will be a minority of examples in which the myocardium is generally good and reference to the clinical picture shows no symptoms associated with heart disease. The existence of such a minority group illustrates the great importance of the *rate* at which the occlusive changes take place rather than the extent to which they may have developed in a given subject. If the rate of occlusive change is slow enough, coronary collaterals may keep pace and take adequate care of the myocardium. If we examine a sufficiently large number of hearts from patients who suffered a painful heart death, there will be a minority in which the areas of coagulation necrosis or fibrosis are not supported by evidence of severe occlusive change and, in occasional instances, the evidence of atherosclerosis in the coronary arteries is minimal. This minority group illustrates the great importance of coronary artery spasm or perverted vasomotor activity of the coronary arterial tree when diseased in one or more of its subdivisions. This brings us to the two current theories for the cause of heart pain. Wood [23] states that Parry first enunciated the idea that relative myocardial ischemia forms the basis of heart pain. It appears likely that if Parry had known that the terminal end plates of the sensory nerve fibers of the heart were confined to the adventitia of the coronary arteries and to the subepicardial surface, he would have never offered this suggestion. This theory was later energetically supported by Sir Thomas Lewis who found it necessary to invoke the idea of a P (pain) substance supposedly elaborated by the ischemic myocardium. The second theory ascribes heart pain directly to perverted vasomotor activity of the coronary arterial tree. I have long been interested in the transient changes in the final ventricular deflections of the electrocardiogram as an evidence of myocardial ischemia. In proper electrocardiographic studies which are correctly interpreted, we have a sensitive and reliable detector of evidences of myocardial ischemia.

I wish to emphatically emphasize that there is a striking dissociation between the electrocardiographic evidence of myocardial ischemia on the one hand and the occurrence of pain clinically on the other. Electrocardiographic evidence of ischemia occurs in somewhat more than half of attacks of angina pectoris. On the other hand, we now know that painless myocardial infarction is not uncommon and there

can be no more severe myocardial ischemia than that which results in infarction-necrosis of great regions of the ventricular wall. Coronary arteries which carry little or no blood may be subject to profound vasomotor change and severe heart pain with little or no electrocardiographic evidence of myocardial ischemia. I will not take time now to labor these arguments further. The trigger mechanisms for the vasomotor attacks of angina pectoris are very important in the diagnosis.

CLINICAL PICTURES

The clinical pictures associated with coronary atherosclerosis are six in number, three are painless and three are painful.

The most common painless form consists of the gradual or abrupt onset of congestive heart failure associated with a rapid arrhythmia which is atrial fibrillation in the vast majority of instances. Here, the rapid ventricular rate fatigues the myocardium and is the direct cause of the congestive failure. The outlook is usually good because the high ventricular rate can be controlled with digitalization. Subsequent to recovery from congestive failure, particular cases may present the problem of converting the arrhythmia to normal sinus rhythm.

The second painless picture likewise unfolds with the appearance of congestive heart failure but is unassociated with a rapid arrhythmia. Under these circumstances, a poorly irrigated myocardium forms the basis of the failure and consequently the outlook is serious and a sharply limited workload (obtained by rest) is invariably required. These subjects are usually over sixty years of age unless hypertension is a component part of the picture. This age period by no means excludes other factors such as "apathetic" hyperthyroidism, or hypothyroidism, and careful consideration should be given to other less common etiological factors.

The third painless picture appears with the onset of syncope in association with Stokes-Adams attacks. The attacks are less frequently due to recurring complete heart block and are more commonly produced by an inadequate ventricular pacemaker following the development of complete heart block. When the inadequate pacemaker is in the form of ventricular tachycardia, quinidine and procaine amide are contraindicated because of the likelihood of ventricular standstill with termination of the attack. Isopropyl norepinephrine has been reported as useful.[24]

ACUTE MYOCARDIAL INFARCTION

Let us next consider some of the problems associated with acute myocardial infarction. One of the difficult ones in this area is prognosis. The family history of heart attacks has been emphasized as unfavorably influencing the long-term outlook. The mortality rate in acute myocardial infarction is about 25 per cent. Of subjects who die in the first attack, 80 per cent die in the first twenty-four hours, 60 per cent in the first two hours and one-half in the first fifteen minutes. In man, the mortality rate increases with age. Thus, in men aged 44 years or under, the mortality rate is low or in the neighborhood of 3 to 5 per cent. By contrast, the rate at 65 years or over is about 35 per cent. In women, the mortality rate is 25 per cent at any age. There are, in addition, a number of complications which profoundly increase the mortality rate. The development of complete heart block is associated with a mortality rate upwards of 90 per cent. The occurrence of acute heart shock carries a mortality rate of 80 per cent; the development of paroxysmal tachycardia of any type. including flutter, carries a mortality rate of 70 per cent; that of congestive failure a mortality rate of 60 per cent; and that of pneumonia a mortality rate of 50 per cent. Wood[23] points out that there are an additional number of findings which tend to be associated with an increased mortality rate. These are a pulse pressure of less than 20 mm. of Hg., a systolic pressure of 90 mm. of Hg., or less, the occurrence of either left or right bundle-branch block, a definite increase of heart

size, the occurrence of intractable heart pain, and finally, the occurrence of acute myocardial infarction in the diabetic.

On the other hand, there are factors which appear to have no effect on the mortality rate. These are the previous presence of angina pectoris; the previous existence of hypertension; absence of heart pain, atrial or ventricular ectopic beats; the location of the infarct and finally, the development of fibrinous pericarditis.[23]

I would like to emphasize certain features in the management of acute myocardial infarction. It is important that the patient receive early skilled medical and nursing care. Vasopressor drugs are required for treatment of acute heart shock. Here, 10 mg. of L-norepinephrine may be added to a liter of 5 per cent glucose and given intravenously through a polyethylene tube at a rate which maintains the blood pressure at 110-120 mm. Hg. in previously normotensive subjects or at higher levels in previously hypertensive subjects. The intravenous or the intramuscular use of heparin in the early days is probably useful even though more prolonged anticoagulant therapy is uncontemplated. An 800 calorie diet with 0.5 gm. of sodium per day is desirable, at least through the first week. It is important to detect the first signs of congestive failure and to treat this complication vigorously. Likewise, the early detection and antibiotic treatment of pneumonia are imperative. The serious arrhythmias must be treated intelligently and without hesitation using sufficient doses of the appropriate drugs. The diabetic with acute myocardial infarction must be managed with a view to avoiding even short periods of hypoglycemia. Antiprothrombin drugs are recommended either in every case or in cases having one or more of the serious complications cited earlier. Generally, when a program of this kind is well carried out, the mortality rate can be reduced by 50 per cent or more.

ANGINA PECTORIS

Angina pectoris frequently commences abruptly but in a given subject usually takes on a stable pattern for a variable period of time extending from a few weeks to several years. The associated heart pain is continuous and of growing intensity. The distribution is midsternal, pectoral, or cervical. It may involve either shoulder and radiate down either the lateral or the medial aspect of one or both arms. At times it may involve the lower jaw. It may be strictly confined to one of the sites of usual radiation or the radiation may be centripetal. The duration is brief, being a matter of several minutes, and is rarely as long as fifteen or twenty minutes. The pain is most commonly triggered by exertion or on emotional upset. Exertion after meals or with exposure to cold are very apt to trigger the attack at a lower level of activity. The chest is symptom-free between attacks and the left submammary region is not involved. Nitroglycerine affords prompt relief; the drug action commencing within thirty seconds. It is important to use the nitroglycerine to prevent, as well as, to stop an attack. The patient should understand that nitroglycerine is not a narcotic, is not habit forming and can be used freely for long periods of time, if necessary.

IMPENDING INFARCTION

There is a clinical picture lying between that of angina pectoris and acute myocardial infarction which is probably based on an accelerated phase of coronary atherosclerosis. The previous pattern of the anginal attacks has changed. The attacks may be more frequent or they may last longer. More characteristically, they begin to occur at rest or may occur at night and thus awaken the subject from sleep. The nitroglycerine requirement is increased and may actually fail to give complete relief. I prefer to call this picture one of *impending* infarction. In this group of subjects we have routinely encountered electrocardiographic evidence of local ventricular ischemia. The location of the ischemia almost always predicts the location of subsequent myocardial in-

farction in those instances when subsequent infarction cannot be avoided. At the present time, I believe it is best to admit these patients to the hospital for a period of two or three weeks whenever feasible. During hospitalization, dicumarol is started and the dose determined which will elevate the patient's prothrombin time to 2.5 or 3 times that of the control. The vasodilator action is usually associated with relief of the increasingly troublesome attacks. The subject is ordinarily back at the previous work level within six weeks after commencing hospitalization. Anticoagulant therapy is continued for at least six and preferably twelve months. In a disease as phasically slow in acceleration or in coronary collateral adjustment as atherosclerosis, it appears unlikely that more than transient benefit might come from shorter periods of antiprothrombin medication. Aside from this argument, there are indications that acute myocardial infarction may be avoided in an appreciable number of instances. Moreover, in the instances when infarction occurs, there is growing evidence that the course is likely to be less complicated and the mortality rate thereby lowered.

I have by no means completely covered the subject but would like to leave the few remaining minutes for questions which I will attempt to answer.

Dr. George Anderson, (Lake Charles, La.): Dr. Bayley, do you think that nitroglycerine is dangerous in lowering blood pressure when the attack of pain turns out to be acute myocardial infarction?

Dr. Bayley: No! I think this point has been greatly exaggerated.

Dr. Anderson: Dr. Bayley, if a patient in an attack of heart pain shows a rather striking increase in blood pressure, would you recommend the use of drugs to lower the blood pressure?

Dr. Bayley: No, I would not use an antihypertensive drug under these circumstances but would use a strong sedative and/or a narcotic if necessary to relieve the pain. In the event that the attack heralds the onset of infarction, the antihypertensive drug might aggravate or help initiate the complication of heart shock. I would like to hear what Dr. Edgar Hull's opinion is in this matter.

Dr. Edgar Hull: I agree with Dr. Bayley.

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REMARKS ON THE REHABILITATION OF THE CARDIAC PATIENT *

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NEW ORLEANS

Discussion of the rehabilitation of the patient with disease of the heart must be divided under two categories:

1. Physical rehabilitation including all measures designed to preserve *cardiac* efficiency so that the patient may again become in some degree a physically functioning unit in society.

2. Psychologic rehabilitation including all measures designed to preserve *personality* efficiency without which the patient cannot again become a functioning unit in society.

I shall confine my remarks to the second. There is no doubt in my mind that more persons with cardiac lesions are invalided and, yes, ultimately destroyed by psychologic forces than by diminution of cardiac efficiency.

Because we shall not have the time to discuss this problem in relation to each kind of heart disease separately and because most aspects of the problem are common to all, I shall choose as a vehicle for my remarks, the cardiac patient who represents both the most common problem in my experience and the most difficult of management. This is the patient who has had a coronary thrombosis.

FEAR

The most powerful force arrayed against us in this situation is *fear*, and it is necessary to speak not only of the patient's fear but of the *family's* fear and last but not at all least of the *physician's* fear.

The patient's fear raises two intolerable spectres: one the threat of imminent sudden death, the other the threat of invalidism, and the first contributes to the second, for in his fear of the grim reaper he is apt to keep himself immobile (under the delusion that even the slightest activity invites immediate dissolution). In this dilemma the members of the family are frequently of no help. Displaying anxiety, giving the patient frequent warnings when he shows any inclination to activity they shatter the weak foundations upon which early in his career as a "cardiac" the patient is attempting to erect the confidence that he needs. The physician, succumbing to the same fears for the patient's continued existence frequently tends to emulate the family's behavior and one cannot help but feel that an important factor here is his fear of the great responsibility involved if one is to attempt to overcome the oversolicitousness of the family; to encourage activity when the family is fearful of it. After all if the patient dies, he fears criticism will appear to be justified.

IMMEDIATE MEASURES

The rehabilitation of the patient who sustains a coronary thrombosis begins with the inception of the attack. Confidence in the physician is of utmost importance and is maintained more by consistency than by his professional status. Accordingly, the patient should be told that he has had a heart attack and the nature of the attack should be explained to him; but he should be assured that with some weeks of rest he will recover and will go back to work and this attitude should not be sabotaged by permitting anyone to tell him that he may not move his arms or legs, that he may not turn over in bed and that he must not worry about anything. Few people are so stupid, even in this benighted world, that the implication of this advice is not clear to them; it not only creates terror but by its inconsistency with the physician's optimistic predictions for the future destroys confidence in the physician and in his methods.

I cannot believe that simple movements

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of the body can be harmful. At the worst certainly they cannot be as harmful as the emotional effects induced by the warning that the patient must remain immobile. Finally I believe that such motions help to prevent thromboembolic phenomena and the shoulder hand syndrome. I actually require that patients exercise the hands and feet after the second or third day in most cases.

No more silly advice has ever been conceived than that represented by the admonition: "You must not worry and you must not think about business or your job and you must not get excited". In the first place, I cannot imagine anything that can create more anxiety for the patient than the notion (implied in such advice) that his heart is so fragile that it will disintegrate if he becomes worried or if he becomes excited. The patient knows, even if the physician does not, that such matters are beyond his control, that worry and excitement are unavoidable. In his attempts to follow such advice the patient suppresses his feelings and thus tension is generated. It is far better to encourage the patient to express his feelings for it is the emotional force that is retained within that does damage within, not that which is disgorged by expression.

Therefore, as early as possible in the attack—on the first day if the pain is relieved and there is neither shock nor heart failure—the patient is permitted to speak with those associated with his work or business so that he can relieve his mind of the responsibilities connected with any unfinished or pending projects. Thus anxiety regarding "matters that should be attended to" is minimized.

The senseless use of oxygen where it is not needed is another factor which impresses upon the patient the notion that he is close to death.

The following days, so frequently uneventful, should be treated casually insofar as the physician's and other attendants' demeanor is concerned. It is important for a physician to be able to make close and detailed observations without the patient's knowledge and certainly without wearing an expression and manner of deep concern. Radio should be furnished almost immediately for amusement.

After seven to ten days, in the uncomplicated case, the patient should be propped up in bed and made to feed himself. At this time he may have television, although watching boxing matches and other sports are prohibited for the time being. He is encouraged to read.

GRADUAL RESUMPTION OF ACTIVITY

After four to six weeks he is ambulated. At this time activity is automatically limited by the weakness in the legs and lack of equilibrium. He is urged to get about and when he can negotiate the distance to the bathroom the greatest single step in morale building and rehabilitation has been achieved. There is no greater, no more deeply significant symbol of human dependence, inadequacy, and indignity than that represented by that onerous contrivance, the bed pan.

From here on the patient is activated as rapidly as possible. At two and one half months or earlier, if possible, he is encouraged to return to clerical or executive occupation on a part time basis. Usually the longer the period of inactivity the more apprehension and tendency to invalidism occurs. Certainly nothing is as completely rehabilitating to the patient's morale as the successful return to his work.

The family is warned to refrain from annoying the patient with a multitude of don'ts. He *may* stoop down, he *may* tie his own shoe laces. He *may* lift a window sash. He *may* move a chair. He *may* climb stairs albeit slowly. He *may* worry. He *may* get excited. He *may* indulge in sexual relations. If he becomes angry he should say just what he thinks. He does not need to go to bed at any special time. He can rest at mid-day or for an hour before dinner.

SEXUAL ACTIVITY

The problem of sexual activity is all too frequently neglected. I make it a

point to discuss this subject at the time that sexual activity should be resumed. In uncomplicated cases this is at two months. Not only does excessively prolonged abstinence give rise to powerful unconscious as well as conscious tensions but also it has been my observation that most persons place great store by their sexual prowess and it is certainly true that most men who are not performing this function at adequate intervals have a feeling of inadequacy which spreads to all areas of activity and self appraisement. In addition there are guilt feelings toward the marital partner. The same applies to most women. Thus, I consider that one of the most important ways in which we can build morale is by encouraging the resumption of sexual relations.

Unfortunately sexual activity along with almost every other activity which gives pleasure has fallen into disrepute. We shall not have time to pursue all of the ramifications of this subject. I must say that with rare exceptions my patients have not had difficulty during or as a result of sexual relations. Two or three patients with the anginal syndrome have had to use nitroglycerine with occasional intercourse. Only one patient had to use it every time.

Apparently the notion is prevalent that sexual relations cause a great strain on the heart. While few scientific observations have ever been made on this subject experience with cardiac patients does not support it. Sexual relations do not seem to be excessively dangerous unless carried out with somebody else's wife.

EMOTIONAL ADJUSTMENT

There can be but one way in which an individual with an unalterable lesion can live a satisfactory and useful life. He must learn to do that which he must do to take care of himself as a matter of habit, and aside from this he must live every day as if he were going to live forever. Patients who are taught to be continually anxious about themselves, to live in unrelenting expectation of sudden disaster cannot function well and cannot find life good. Every step and every attitude that I have described here is calculated to bring about a pattern of emotional adjustment at the healthiest possible level. Failure to do this results in much anxiety and this leads to pains in the chest that are mistaken for cardiac pain and all too often lead to further restriction and more anxiety and more invalidism. Here the physician is in a most difficult position. It is extremely difficult to know whether pain in the chest in such cases is due to cardiac events or not. Clinical judgment and electrocardiographic know-how are essential. It is necessary to realize too, that the effect of concluding that the pain is cardiac when it is not is just as harmful as the effect of concluding that it is not cardiac when it is. Physicians who cannot stand the responsibility of such decisions, who must always do what they think is "playing it safe" should refer these patients to those who do not feel so insecure.

Necessarily the greater the damage sustained by the heart in this as well as in other cardiac disease the greater will be the difficulty encountered in maintaining cardiac efficiency and the greater will be the problems of psychologic adjustment. However, the same principles should be followed. Whatever medications he must take, whatever restrictions he must follow are to be made a matter of everyday routine but with the attitude that it is given to no one to know how long he will live and that he may as well live as if he is going to live forever. To accomplish this it will be necessary for the physician to know his patient well, to have long sessions with him, airing his anxieties, and his misconceptions, and frequently dealing with some of the personality difficulties that most of us have, and with whatever religious or philosophic resources the patient may possess.

Of course, not all patients can be rehabilitated and this applies to the psychological as well as physical rehabilitation. However, I have many patients who have been working for periods up to more

than twenty years following a myocardial infarction. Some have angina, some do not. Some have had two myocardial infarctions and some are taking mercurial diuretics regularly to prevent congestive heart failure.

WORK

Most of these do clerical or professional work. Some are artisans. None do extremely hard work.

. The objective of rehabilitation in most persons with heart disease can be attained if proper attention is given to both the physical and psychological problems encountered. The patient who is self-employed is fortunate. But the patient who is not, frequently furnishes us with some of our more frustrating and tragic experiences. He has been taught that he can work and we are responsible for this teaching and when an examining physician for some industrial plant tells him that he cannot be hired he is left with precisely the problem he has feared and the physician is in the position of deceiver. The latter point is not made through preoccupation with the physician's personal problems. The importance of this point here is that the feeling that his physician has deceived him adds a great deal to the patient's demoralization, the destruction in a moment of weeks of morale building.

I do not know of any quick solution for this problem. Close cooperation between examining physicians and the patient's physician may do much to soften the blow of rejection but this is hardly our objective today. These people are employable. Necessarily, some jobs must be closed to them, particularly those that involve hazards to other persons. However, most of them who have not been doing very hard manual labor can go back to the jobs they left, some possibly on a part time basis. I believe that industry, given proper understanding by the profession will cooperate. But it seems to me that the extent to which industry can cooperate is limited by some of the provisions of the liability laws. Among the working public whose jobs come under the exercise of these laws is a large group of people who have hypertension, who have heart disease and who are *going* to have hypertension and/or heart disease. It behooves them in cooperation with industry to seek to make such provision in the laws that will make it possible for industry to protect the cardiac's opportunity to earn a living without assuming any more than its proper share of the responsibility for what happens to the person on the job. Then more cardiac patients will be permitted to work, will feel useful, will be happier, and undoubtedly will live longer.

"A light heart lives longer"—(*Shakespeare*).

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PREVENTION AND MANAGEMENT OF HEART FAILURE: GENERAL PRINCIPLES AND THEIR APPLICATION *

HAROLD J. JACOBS, JR., M. D.
LAFAYETTE

Few other disease states have received the interest and respect of the practicing physician and the research laboratory as has the condition of heart failure. It is paradoxical that treatment is for the most part quite effective, yet much of the pathologic physiology is unknown.

It is impossible to categorize completely the prevention and management of heart failure, for many of the principles are applicable in both instances. Likewise, the treatment of the varying degrees of failure—mild, moderate, severe, intractable, acute or chronic—is similar in principles, the vigour with which it is approached being related to the severity, to the acuteness, and, as is sometimes overlooked, to the etiology of the failure.

PREVENTION

The best and most desirable method of preventing heart failure is the prevention of heart disease. This is an obvious and rather silly-sounding remark, but I make it with good intent. I would like to take advantage of my position today to stress

* Presented at meeting of the Louisiana Heart Association, May 15, 1957, in Shreveport, La.

the importance of early, adequate and prolonged treatment of all hemolytic streptococcal infections and the continued prophylaxis against infections of this type in individuals who have had rheumatic fever. Even though this plan of attack has received widespread publicity in the medical literature, many patients are still not being treated in this fashion. I realize that all of you here today use the treatment recommended by the American Heart Association,[1] but I will reiterate the principles briefly—treatment of streptococcal infections with therapeutic doses of antibiotics for at least ten days and prevention of streptoccal infections in rheumatic individuals for life by a monthly intramuscular injection of bicillin or daily doses of sulfadiazine or oral penicillin.[2]

An excellent example of what can be done in the prevention of heart disease is typified by syphilitic heart disease. A mere ten years ago, this was a common entity, particularly on the wards of Charity Hospital. Today, it is a comparative rarity, thanks to early and adequate treatment of syphilis.

The question of preventability of coronary or arteriosclerotic heart disease has received considerable attention in both the medical and lay press lately. From the evidence at hand, it seems that diet, and particularly a high fat diet, has been incriminated as the causative factor. Prevention of coronary disease in a susceptible population by elimination of fats in the diet remains to be proven.

Prevention of hypertensive heart disease meets with its greatest success in those instances in which the hypertension can be cured, not just temporarily alleviated, by surgical means. Examples of this are coarctation of the aorta, pheochromocytoma, and unilateral renal disease.

There are other forms of preventable heart disease, but time does not permit their receiving consideration.

In the presence of pre-existing heart disease, much can be done towards the prevention of the development of heart failure. The rapid strides in cardiac diagnostics and in surgical techniques in the last ten years makes this particularly true. Examples of surgical correction of heart disease are familiar to all of you—mitral valvulotomy, pulmonary valvulotomy and infundibulectomy, closure of septal defects and ligation of persistent ductus arteriosus, to mention just a few.

The problem of pregnancy in heart disease bears special consideration. Pregnancy should be avoided in certain cases. It is true that the maternal mortality rate is only slightly increased in rheumatic heart disease[3] and that interruption of pregnancy is rarely indicated for cardiac reasons; however, interruption of pregnancy should be *considered* in the presence of tight mitral stenosis, increased pulmonary pressure, active rheumatic fever, subacute bacterial endocarditis and increasing hypertension.[3, 4] If interruption becomes necessary, it should be done as early as possible after compensation has been restored, but usually should not be attempted after the sixth lunar month. Termination of pregnancy before term to avoid the strain of labor is unnecessary since heart failure rarely develops during labor.[5]

There are many other things which should be done in the presence of heart disease to prevent heart failure. The activity of the patient should not exceed what the heart is capable of taking care of, so emotional and physical strain should be limited, especially in coronary and hypertensive heart disease. Respiratory infections should be avoided or treated early. Chronic cough should be controlled, for the strain involved may precipitate failure. Rapid or excessive intravenous fluid administration must be avoided, particularly with respect to sodium-containing solutions. Obesity should be reduced, since this causes extra work for the heart. Master et al have reported a 35 per cent reduction in cardiac work by the loss of 12 to 15 per cent of body weight in obese patients.[6] Of course, along with dietary management, it may be necessary to restrict sodium intake.

Then there is a group of conditions which may cause heart failure without the presence of primary heart disease. These are arteriovenous fistula, anemia, beri-beri, hyperthyroidism, hypothyroidism, and pulmonary disease. Correction of these diseases will prevent heart failure.

So much for prevention.

MANAGEMENT

In the management of heart failure, the first thing to be done is to make sure the patient has heart failure. The things most likely to be confused with it are pulmonary disease, anemia, and constrictive pericarditis. After one has established the fact that the patient has heart failure—at this point we could, but will not, get into the very controversial subject of "what is heart failure"—and before treatment is begun, an accurate diagnosis should be made. By this I mean an accurate etiologic, anatomic, physiologic, and functional cardiac diagnosis as outlined by the New York Heart Association.[7] Such a diagnosis will dictate in large part what should or should not be done. The objectives of treating heart failure are twofold: first, to treat the failure per se and, second, to treat the underlying condition which has caused the failure.

In some instances, the underlying condition must be treated before the failure can be controlled. An example of this is failure resulting from longstanding paroxysmal auricular tachycardia. Stopping the tachycardia by tricks of vagal stimulation, such as carotid sinus pressure, eyeball pressure or pressure on the supraorbital nerve, usually results in rapid recovery from the failure. Other examples such as this are thyrotoxicosis and severe anemia.

The following general principles of treatment are not listed in the order of their importance or in the sequence in which they should be used, for this will differ from one case to the next.

The patient that presents himself with heart failure is usually quite apprehensive, particularly in the acute form. Mental rest is important, for agitation increases the work load of the heart by increasing blood pressure and pulse rate, so make every effort to relieve the apprehension. Be optimistic. In the long-term care, keep the sickroom pleasant, quiet and as cheerful as possible. Restrict visitors, and this should include aunts, uncles, cousins, grandchildren, etc., who usually say they are not visitors but are part of the family. The patient should not be allowed to conduct business from the bed. This may be modified in some instances, for the worry about the business may be worse than the work involved in making temporary arrangements.

Physical rest is probably the most important part of the management of heart failure. In many instances rest alone will control the symptoms and signs. In heart failure, cardiac output is insufficient to meet the demands of the body, but this output may be adequate at rest. The failing heart is unable to increase its output in proportion to increase in activity. This leads to inadequate blood flow to the kidneys, to increase in venous pressure, to increase in venous return to the heart, and in left ventricular failure, to pulmonary congestion and dyspnea. On the other hand, rest improves cardiac efficiency by decreasing blood pressure as a result of lower peripheral resistance, by slowing the heart rate, allowing a longer period of diastole or rest for the myocardium, by less venous return, and by less oxygen requirement, which tends to decrease dyspnea which in turn cuts down on the muscular effort of respiration.

Even the question of rest is a controversial one. What is the best position for rest in failure? The dyspneic patient feels that he must sit up in bed or at least be propped up on several pillows, but is this a restful position? It has been advocated that blocks be put under the head of the bed in order to incline the whole bed at an angle of about 30 degrees.[8] This is better than elevating just the upper segment of the bed. On the other hand, the sitting posture in a chair in the treatment of failure has very strong proponents.[9] This decreases venous return to the heart

by pooling of blood and by immobilization of edema fluid in the lower extremities.

What degree of rest should be ordered? Theoretically, it should be absolute; however, absolute bed rest has disadvantages. It tends to emphasize the seriousness of the situation and may make the patient more apprehensive. It may cause constipation, abdominal distention, bed sores and even osteoporosis if prolonged. It is best to allow the patient bathroom privileges or the use of a bedpan. Frequent moving and deep breathing is also indicated to prevent the development of pneumonia and thromboembolism.

How long should rest be continued? There is no definite answer to this question for the ideal duration of rest has not been established. Some feel that bed rest should be prolonged in order to let the heart recover. Obviously the etiology of the failure has a great deal to do with this question. If the failure is secondary to a recent coronary occlusion or to activation of rheumatic fever, the period of bed rest should be longer than if the failure alone were being considered.

The use of oxygen has long been recognized as being of value in the treatment of heart failure, though in the majority of instances, arterial oxygen saturation is normal. This may even be true in the cyanotic patient where the cyanosis may be due to stasis of blood in the skin from elevated venous pressure rather than from actual anoxemia; however, oxygen should be used since most patients experience subjective improvement from its use, since it reduces the work of breathing, and since it is insurance against oxygen lack, to which the failing heart is particularly susceptible. In the presence of pulmonary edema, oxygen under a pressure of 5 to 8 mm. Hg. may be of considerable benefit, increasing oxygenation of arterial blood and even more important, decreasing venous return to the heart, helping to clear up the pulmonary edema.[10]

Diet is very important in the management of heart failure. During digestion and assimilation of food, cardiac output may increase as much as 30 per cent and this increase in work may last two to four hours.[11] Fortunately most patients are not interested in food for the first few days. When the patient starts to eat, some degree of undernutrition is aimed for, for this results in a decrease in metabolic rate, lower cardiac output and lower blood pressure and pulse rate. This type of diet is particularly indicated in the obese patient. Undernutrition also increases the vital capacity and tends to induce diuresis.[12] Small frequent feedings are given to prevent gastric filling which would decrease vital capacity. A bland low-residue diet is used to prevent distention.

Reduction of sodium intake is the most important part of dietary management since the retention of fluid and increase in blood volume are dependent upon sodium retention. A patient in failure should not be allowed more than one gram of sodium chloride a day to begin with. This may be achieved with the Karrell diet, which consists of 800 cc. of skimmed milk only per day. More rigid salt restriction may be found necessary. In such a case, some may find the diet recommended by Olmstead et al[13] to their liking. This diet consists of 1150 cc. of beer daily. It supplies 480 calories, no salt at all and is an excellent diuretic. Let me emphasize at this point that it is extremely important to keep in mind the amount of sodium contained in the patient's water supply. It imposes difficult and rather futile restrictions to put a patient on a diet limited to 200 mgm. of sodium per day and then to overlook the fact that he may get more than this amount from the water he drinks. In order to make a low-salt diet more palatable, salt substitutes may be used, but since some of these contain potassium, they should not be used in the presence of severe renal disease.

Water restriction is not necessary, even in the presence of edema, for when on a low-salt diet, water acts as a diuretic. The patient should be allowed water as his thirst dictates and should not have it forced upon him.

Since one of the pathological states found in heart failure is increased blood volume, the rapid removal of 350 to 1000 cc. of venous blood is often dramatic in its effect and oftentimes life-saving. Almost the same thing may be accomplished by the application of tourniquets to the four extremities. These tourniquets should be applied as close to the trunk as possible and tight enough to occlude only the venous return. The hazard of phlebothrombosis with this procedure must be kept in mind. Phlebotomy may be very effective in left ventricular failure but is rarely of benefit in right ventricular failure secondary to pulmonary disease. Obviously a phlebotomy should not be done in the presence of anemia.

The accumulation of edema fluid and serous effusions makes it difficult for the body to get rid of its excess sodium and water. In addition, pleural effusion may be a mechanical interference with respiration. When this occurs, removal of the fluid by thoracentesis is indicated. Recently, the pleural space has been likened to a sump, with the recommendation that all pleural effusion, regardless of its effect on respiration, be withdrawn as a further means of relieving the body of salt and water, and that this removal of fluid from the chest be continued as long as it continues to appear.[14] Abdominal paracentesis for the presence of ascites usually is not indicated unless there is marked distention and unless it does not respond to treatment. Subcutaneous edema usually disappears with the control of heart failure and only rarely is the use of Southey tubes necessary.

Many surgical procedures have been used in the management of heart failure. Though a functional class four patient is not an ideal candidate for a mitral valvulotomy, this procedure may be a life-saving measure. In chronic heart failure which is difficult to control, complete thyroidectomy has proven of some benefit. There are times when surgery is a must, for example, decortication in constrictive pericarditis (though this is not true heart failure) and closure of the fistula in arterio-venous aneurysm. One operation which as yet has not received much favor in this country for the control of heart failure is ligation of the inferior vena cava. This is usually reserved for cases of intractable failure and is said to be particularly indicated in mitral and combined mitral and aortic disease.[15] Improvement is reported to be immediate, but mortality high. Where persistent heart failure is due to recurrent pulmonary emboli from the lower extremities or pelvis, inferior vena cava ligation is the treatment of choice.

Now, what about the little luxuries that the patients usually ask about when they begin to feel better—the question of the use of tobacco, alcohol or coffee. In *normal* man, cigarette smoking causes an increase in peripheral resistance, a decrease in venous return and a decrease in minute output.[16] It also causes a significant rise in coronary blood flow and heart rate and a significant decline in coronary vascular resistance and myocardial extraction of oxygen and glucose.[17] Let me emphasize that these effects are in normal man. The EKG changes caused by smoking in the presence of coronary artery disease are well known. Smoking has no direct effect on heart failure, however, it may aggravate the situation by causing coughing or palpitation, to say nothing of the effects of the carbon monoxide inhaled. Coffee or tea is allowable in small amounts, though they may cause nervousness, irritability, and even palpitation. Remember that they are good diuretics, having much the same effects as aminophylline. There is no contraindication to small quantities of alcohol.

During the convalescent period, the heart failure patient should be returned to activity with gradually increasing exercise. Modified bed rest should be continued until recovery seems complete. These patients should be encouraged to return to work, depending upon their functional classification, even if it is necessary to modify their work according to their tol-

erance. A study by Goldwater et al shows that employment has no harmful effects and that progression of heart disease is related more to age than to employment.[18]

Unless the episode of heart failure was due to some temporary and transient etiology such as infection or anemia, the same principles, in some degree, of the prevention and management of heart failure must continue to be applied in the long term care of the patient with heart failure.

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PREPARATION FOR LARGE SCALE INFLUENZA EPIDEMIC

It is the considered judgment of those who have access to adequate epidemiological and virological information that there will probably be an outbreak of a rapidly spreading type of influenza in the United States in the coming fall and winter. It is anticipated that in addition to the usual amount of influenza there will be outbreaks of the oriental type of the disease recently prevalent in the Orient. According to reports from the Public Health Service, 13,000 confirmed cases have been reported in this country up to the middle of August.

Influenza epidemics have apparently occurred in all ages past and there is a tendency for intensification of influenza to appear at intervals of about twenty years. Epidemics of influenza started in northern China in January of this year, and have appeared successively in Hong Kong, Singapore, Taiwan, the Philippines, the Malayan States, Indonesia, Japan, and India, and more recently, in this country. This indicates that a recrudescence of the disease may be reasonably anticipated in the near future.

The problem was recognized by physicians in the U. S. Army medical service, and studies of the epidemiology of the disease were undertaken in various laboratories, particularly the Army's 406 Medical General Laboratory in Japan. It was established there and in other laboratories working at the same problem that a new strain of type A influenza virus has appeared. Because it is a new strain there is no natural immunity. On May 22, prototype strains were sent to vaccine manufacturers. Preparations for the production of a suitable vaccine were undertaken. It was planned that 60 million ccs. would be available by February 1, and probably sooner, and that some lots would be ready for distribution after the middle of August.

Clinical information regarding the Oriental influenza indicates that the attack is similar to the type physicians are familiar with. The incidence and attack rate averages approximately 20 per cent with various reports running from 15 to 50 per cent. The incubation period is probably one to two days, and the contagious period uncertain, one to five days. It is transmitted as is other influenza by droplet infection. The symptoms of fever, prostration, headache, and lassitude develop rapidly, fever varying from 100 up to 105. Symptoms are accompanied by coryza. The course is three to five days. Complications are said not to be serious, except in the very young and in those debilitated. The mortality is low and said

to be in the Orient 0.2 of 1%, but in this country only three deaths have been reported.

The treatment recommended is similar to that familiar to physicians generally, with the added statement that the antibiotics, while of little value in affecting the disease process provoked by the virus, are of substantial value in protecting against secondary infection. The treatment thus becomes supportive and protective.

The brief duration of three to five days is usually followed by a period of prostration or weakness lasting probably twice as long as the fever.

The prospect of any disease process, even though short in duration, affecting 20 per cent or more of the population, is one which would properly give physicians concern. The president of the A.M.A., Dr. David B. Allman, has stated the epidemic may never strike this country but the American Medical Association is anxious to have practicing physicians mobilized and alerted in case an outbreak does occur. The Board of Trustees has set up a special committee on influenza to implement the informational and operational phases of the program which the A.M.A has proposed. It is felt that the American Medical Association can provide leadership to acquaint state and county medical societies, as well as the general public, of the situation. The rapid onset of influenza of this type makes it necessary to consider the problem well in advance and to encourage the development of plans prior to any massive outbreak. Articles in the Journal of the A.M.A. will appear providing information on the clinical aspects and current information concerning the disease. Public information will be provided by the Public Relations Department.

It is urged that state and county medical societies prepare or develop adequate stand-by programs and plans to cope with such epidemics as may develop. The Orleans Parish Medical Society has undertaken to organize a suitable program, and it is learned through the Washington News Letter that Mississippi has already laid out a complete statewide emergency program. It is stated that consideration should be given to the provision by which expanded professional care could be gained through utilization of all medical personnel regardless of type of practice. Hospital facilities should be made available through curtailment of elective surgery and diagnostic studies, and so forth. The vast majority of patients will of necessity be cared for in their homes. The need and possible scarcity in the handling of a massive epidemic would be one of medical supplies, patient care, and physicians' services. It may be expected that as usually happens the opening of schools and the onset of cool weather will be followed by a sharp increase in the amount of respiratory infection. Under such conditions, the Oriental influenza infection with a comparatively nonimmune population could reasonably be expected to spread rapidly. The only protection is the use of the special vaccine for this particular strain. The Board of Trustees of the A.M.A. and the Public Health Service have approved a statement which in part says that "the Public Health Service in cooperation with the medical profession will stimulate and promote a nationwide voluntary program of vaccination against the prevalent strain of influenza." There is, however, no plan for the use of federal funds for the general purchase or administration of the vaccine.

Maximum public use of the vaccine is being urged as rapidly as supplies become available. The dose will be 1 cc. A single injection is considered to be about 70 per cent effective, and protection develops in ten to fourteen days. The Public Health Service has urged that particular consideration be given to the vaccination of those whose services are imperative for the care of the sick and those needed to maintain other essential functions.

Not before now has it been possible to estimate even the probable appearance of an epidemic of influenza. The combined efforts of epidemiologists and virologists

indicate strongly that one is approaching. With the knowledge gained from the epidemic of 1917-1918, that a good bed was better than unlimited will power in combatting the disease, and that added assistance can be gained from the use of antibiotics, the epidemic that is probably approaching should give no cause for alarm. However, to minimize its effects careful planning, and energetic pursuit of the immunization program should be undertaken by the physicians.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

REPORT OF FIFTH NATIONAL MEDICAL CIVIL DEFENSE CONFERENCE

The Fifth Annual National Civil Defense Conference held at the Waldorf Astoria in New York was attended in the interest of the Louisiana State Medical Society. The program was sponsored by the Council on National Defense of the American Medical Association and included top men in each phase of the problem, including Dr. David B. Allman, President of the A.M.A. who sounded the following warning to physicians:

"It is apparent that every surviving physician —specialist or general practitioner—from a rural area or a metropolitan center, will have a direct medical responsibility in the management and care of mass casualties in the event of atomic warfare. This is a gigantic task which few physicians fully comprehend."

The committee elected to print the following paragraph at the beginning of its program:

"The ultimate responsibility for the health and medical care of our nation's population, in peace or war, rests on the medical and allied professional groups and they can discharge such responsibilities wisely only if they are adequately informed and equipped. The purpose of this program is to better acquaint members of the medical profession, as well as other medical and health groups, to their responsibilities with respect to the hazards and effects of lethal radiation and radioactive fallout."

Three hundred physicians and national, state and local civil defense and health leaders attended the conference which began at eight o'clock in the morning, and continued through serious and grim discussions until five o'clock in the evening. Everyone present was impressed with the seriousness and sincerity of the speakers and the tragic facts of their respective subjects.

The administrator of the FCDA outlined federal civil defense affairs and plans outlined for their implementation. The details can be found in pamphlets published by the FCDA.

Congressman Chet Halifield of California, Chairman of the Subcommittee on military operations, Committee on Government Operations, delivered a formal address on "Civil Defense —A Program of Federal Responsibility", in which he strongly urged federal leadership and funds to implement a federal program. The following is quoted in part from his plea.

"I shall attempt to discuss the federal program from the legislative standpoint, indicating where we stand today in our endeavor to build a responsible federal program of civil defense.

"The reason for such a program is the evident fact that nuclear weapon technology has far outpaced the techniques of civil defense preparedness. The hydrogen bomb following on the heels of the atomic bomb, and new vehicles for delivery of such weapons promised in the rapid development of long range missiles, create new dimensions for the tasks of civil defense.

"Traditional techniques attuned to conventional weapons and the possibility of isolated attacks on limited areas will no longer serve. The civil defense job today is to provide protection for the entire population. For several years, the official policy has been that distance between yourself and the point of anticipated weapon detonation; in short, to evacuate. As we run out of distance, as the major population centers become exposed to multiple megaton bomb attacks, and the entire country becomes subject to radioactive fallout, then the primary question is one of providing shelters for all the people.

"Adequate shelter is only the starting point of an effective civil defense program. The total task of civil defense against a mass attack embraces the initial saving of lives, the treatment and care of millions of casualties, and the preservation of the means of our very survival.

"Obviously, an important part of the recovery effort will have to be borne by the medical pro-

fession, without encroaching on the technical areas of discussion to be covered by the extremely qualified speakers who come later in the program, I want to congratulate the medical profession for its efforts to prepare for this unprecedented task. This meeting today is evidence of your genuine interest in doing your part in a possible civil defense emergency.

"The magnitude of the total civil defense task facing our country has made a tremendous impact on the members of the house military operations subcommittee, of which I am privileged to be Chairman. Our study and investigation of civil defense led us to conclude that to fulfill its constitutional responsibility for national defense, the federal government must assume a positive leadership in civil defense. The states and localities must be assisted in their own efforts to provide for the safety of their people, and in addition, the federal government must undertake to provide a comprehensive national program within which the states and localities can work.

"One of the obstacles now facing the proposed legislation at this time is that its implementation will cost money. As you are no doubt aware, the present Congress is not in a mood to encourage additional federal expenditures. Not since I have been in Congress have I seen a more economy-minded session. . . . .

"While these administration amendments would permit additional federal contributions to the states for various limited purposes, they would not materially change the basic philosophy of that law which gives (I should say "abandons") primary responsibility for civil defense to the states and localities.

"An executive which has not asserted positive, aggressive leadership in behalf of civil defense, and a Congress bent on slashing appropriations to the bone are not a winning combination for the goal we have set—a new charter for civil defense. Our subcommittee is determined, nevertheless, to do everything in its power to put across this legislative program. . . . .

"One of the compelling reasons why the federal government must accept the major responsibility for civil defense today is that the nature of the hazards confronting us can never be fully understood by civil defense planners in local units of government. The federal government should make full use of the knowledge and talents available in the weapons effects field and devise a comprehensive civil defense program for the entire country.

"Not only is this a constitutional duty of the federal government; it is a practical necessity if we are ever to have an effective civil defense program. Obviously, the success of any nationwide program of this type will depend on strong support and participation by the states and localities. But the starting point must be the formulation of a realistic program and the provision of necessary financial assistance by the federal government.

"For this reason, I appeal again, as I have in the past, to the President and the executive branch to recognize and accept this responsibility. If the executive branch will do the part which it can do best under our governmental system—provide a focus of national leadership and a rallying ground for public support—then the congress, I am sure, will do its part by voting the authorizing legislation and the funds. In the meantime, our subcommittee intends to keep reminding both branches of government—and the public as well—that the civil defense job still remains to be done. . . . . "

Following the most convincing address of Congressman Halifield, Mr. Jack C. Green, Director of Radiological Defense Division Health Office F.C.D.A., Mr. Benjamin C. Taylor, Director Engineering Office, F.C.D.A., and Dr. M. M. Van Sant, Director Medical Care Division Health Office, F.C.D.A., conducted a two hour symposium on "Meeting Radiation Hazards and Delineating Programs and Plans."

The theme that ran through the whole discussion was national survival, as distinguished from the individual care of particular patients. The radiological briefing outlined the whole scope of irradiation from initial damage to delayed effects on human subjects and subsequent effect on animal and vegetable life by fallout stressing the hazards of a possible permanent infestation of animal bone.

The whole subject of radiation effects was discussed in minute detail which would be too lengthy to delineate in this report. The controversy over the possibility of genetic changes in future generations was discussed at length here and later in the program. It was urged that decision and judgment on this subject be reserved until all facts are reported—and in reading reports you are urged particularly to note the background of the individual giving the report and to consider his capabilities and competency to make a report on the particular phase of irradiation about which he might be speaking or writing.

Protection from irradiation was discussed at length. The interposition of various materials between the subject and the bomb were outlined. Reinforced concrete was considered the best irradiation shield, although it was stressed that nearly any substance affords some degree of protection. It was brought out that three feet of earth affords a complete shield and absolute protection from bomb irradiation and fall out.

Dr. Van Sant outlined the gruesome possibility that the United States, following a mass bombing, could sustain a loss of two-thirds of its population. This, he stated, could possibly occur in the 1960's, when the enemy potential would be strong enough to deliver such a blow.

He outlined the return of primitive care of the mass casualties. Most hospitals would be rendered useless or destroyed. Medical men would be scarce with fewer, and in most cases only primitive tools with which to work.

The old barbaric custom of caring for the most seriously injured last would, of necessity, have to be revived. He said "with roughly two-thirds of our population killed or injured one-third relatively uninjured, that first and second priorities for medical care should go to those who could return to work immediately and those incapacitated for a short time. Third and fourth priorities were assigned to those whose recovery will, in time, increase our work force and to those who must have medical care, but which can be deferred.

"Final priority will be given to those who may well be expected to die, regardless of their treatment or to those for whom treatment would mean the denial of survival to higher priority groups, due to time of ministering personnel and the amount of supplies required."

Following this, a symposium on the effects of irradiation was conducted by Dr. Eugene P. Cronkite, Head of the Division of Experimental Pathology, Medical Department, Brookhaven National Laboratory; Dr. Henry Bowman, Chemical Engineer, Dean of Drexel College; and Dr. Cyril Comar, Chief of Biomedical Research, Oak Ridge Institute for Nuclear Studies.

Dr. Cronkite opened his remarks with the observation that there is as yet, no satisfactory solution to civil defense.

He outlined the sources of information that are being studied. Hiroshima and Nagasaki data, the 1954 Pacific Island studies, reactor accidents, and therapeutic irradiation.

He outlined the effects on the body at varying distances from the explosion and the possible survival after exposure to large doses of irradiation. He stated that with an exposure of 200 R or less, survival would be expected, and that it might be possible to survive doses up to 800 R., but that it was impossible to stand 800 R. or more and that death in such case was inevitable.

He touched on the subject of the contamination of food from fall out and the harmful effects of ingestion of contaminated food and water, and breathing contaminated air.

The simple precautions that can be taken were outlined and recommended. Washing the body with soap and water; putting a wet wash cloth over the face to filter air, and sprinkling the dust on the ground with water to hold down air pollution; tin cans as protection for food was considered good. Shelters, he thought, are the best method of protection for all.

Professor Bowman followed with data from the Nagasaki and Hiroshima studies; making the point that the bombs used now are one hundred and more times more destructive than those used at that time.

He showed lantern slides and pictures of the destruction wrought in these areas, and noted the fact from his pictures that anything that acts as a shield gives considerable protection. The slides included a demonstration of the bombing effect on different types of construction. Reinforced concrete walls withstood the blast best, while brick and concrete blocks made a poor showing.

He also stressed the magnitude such a disaster as nuclear bombing would cause and the chaos that would follow the blow. Psychological and primitive medical measures to combat this were sketchily outlined.

Dr. Comar was next on the program and gave a discourse at some length on the effect of fall out on animals and crops, and said that "strontium-90 is most important because it is readily taken up into all phases of biological life." The radioactive iodine was considered less harmful. Cattle can stand 300 to 400 times higher concentrations than humans, and they get 80% of their iodine from forage. "Very minor chances of much harmful activity on the thyroid gland would result." On the other hand strontium-90 goes into the stratosphere and is distributed world wide, where it will get into plants and animals.

The method of estimating strontium-90 is in terms of calcium and one microcurie to the gram of calcium was considered the maximum.

He outlined three phases of the study being conducted, as follows. 1. Survey of the amount of strontium-90 in body, soil etc. This information, he said, is coming in well. 2. Study of the metabolism of these items in the food chain. "Can we increase the calcium intake without increasing the strontium-90 intake? This will take much more work." He interpolated parenthetically that "calcium is absorbed better than strontium-90 and that milk will increase the absorption of strontium-90." 3. "At what level of strontium-90 do we need to worry? There is not much information on this now. Levels as now exist are not thought to cause damage. Much more experimental work needs to be done to determine what is the maximum amount of strontium-90 that we can stand."

Major General James P. Cooney, Deputy Surgeon General, M. C., U. S. Army sketched the military concepts of the medical management of

radiation casualties. He began by disagreeing with other members of the panel as to the total number of mass casualties at one time. He made the observation that we have excellent military defense, and that with the radar warning system to alert the air force the navy and the army, sufficient time should be obtained for the population to either take shelter or be evacuated, while the military should be able to reduce the attacking force by considerable numbers. He placed the top figure as only twenty million casualties!!.

In handling of casualties he put "more emphasis on first aid, self aid and 'buddy' aid" Medical help may be two weeks off, and medical supplies will be curtailed and no luxury treatment can be given.

Man power will be short and he reiterated that the best chance for national survival will be to treat the least injured first. The best surgeons will be used to classify casualties for treatment. "If a soldier is burned on the face and hands and can still carry a gun he still works. The same holds true for the terribly irradiated. They must still fight since they will die anyway."

The subject of protective clothing was discussed; whether uniforms should protect against beta or gamma rays or both. He seemed to think that too much clothing would be in the way.

Dr. Jos. W. Howland, Chief, Medical Division A.E.C.P. followed with the civilian concepts of radiation casualties. "Panic and disorder will be inverse to the time of warning before the bomb hits" were his opening remarks.

He outlined the different sensitivity of the various organs and tissues of the body to irradiation, and stressed the variations of the irradiation syndrome, likening it to **aplastic** anemia. He also observed that children and the aged were more susceptible to irradiation than the mid-life age.

Decontamination of casualties was discussed. "All casualties must be screened before moving and clothing must be disposed of. Exposed areas must be washed and all hair clipped off." Personnel working among casualties should be evacuated after an exposure time equal to fifty-R.

Treatment of casualties was delineated. Washing the body with bland soaps for decontamination, and supportive treatment for a two weeks period. Antibiotics can be given after one or two weeks if available. He observed in passing that the efficaciousness of tetracycline was not proven. Control of hemorrhage was mentioned along with its prevention, if possible.

Transfusion for anemia if available, but "in the main medication should be simple and oral. Little if any laboratory facilities will be available or needed."

Prophylaxis: Early warning, the fall out should be visible allowing evacuees to find clean areas and any form of shelter, which will reduce the dose to about half. "Three feet of earth covering reduces hazard by 1/5000 and you should remain protected for 36 hours. Self help is essential."

This concluded the symposium on radiation and radioactive fall out.

Colonel Francis B. Stewart, U.S.A.R. Consultant, Chemical and Biological warfare defense F.C.D.A. presented a film entitled "Treatment of Nerve Gas Casualties."

Experimental animals were used to demonstrate the rapidity of onset of symptoms and eventual death, unless the premonitory signs were recognized early and prompt treatment instituted.

It was stressed that the inhalation of only a small amount of the gas is necessary to produce disastrous results. Symptoms are present within two minutes. Weakness and lack of coordination are observed early. Frontal headache, pains in the eyes, contracted pupils, inability to focus the eyes, tightness in the chest, labored, distressed breathing, nausea, profuse perspiration follows, and finally convulsions and prompt death completed the picture.

The treatment must be rapid and energetic. Two milligrams of atropine intravenously repeated at five to ten minute intervals for three doses; followed by more oxygen if necessary. Recovery is prompt when treatment is instituted early.

The conference was closed by Dr. Cortez F. Enloe, Jr., member Committee on Civil Defense and Council on National Defense, AMA, who stressed the salient points made by earlier speakers.

He warned particularly about the widespread propaganda to prevent further atomic tests, and of the fallacious nature of sentimental judgments. He particularly warned about passing judgment on the ill effects of fall out before complete knowledge is available, and to "only take facts from known specialists in each field. We need more scientific data and knowledge from further tests. Each test gives new knowledge."

This brought to a close a day-long portrayal of the melancholy plight mankind will find itself confronted with unless international gangsterism can be suppressed, and the communist nations of the world returned to freedom with a peaceful coexistence under God.

William E. Barker, Jr., M. D.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

A FEW WORDS FROM PRESIDENT EISENHOWER

"There is one basic thing to remember about hiring workers who are physically handicapped. It is good business to hire them—good for the nation and good for the person. It makes an earner out of an American who would otherwise be relatively helpless. I would like to congratulate the citizens who understand this and are helping to promote the widespread use of handicapped workers.

"Now we must tell others about the value of employing the physically handicapped because two million Americans with physical handicaps are still waiting to be used. Two million Americans could enter the labor force today if they were properly prepared and equipped to do so.

"I urge all employers, therefore, to use the handicapped wherever possible. I urge all workers to accept their handicapped fellow Americans as their co-workers. In these demanding times the labor force of our nation is our most precious asset. Working shoulder to shoulder, the handicapped can add spirit and power to America as we seek to promote the strength of the whole free world."

NEW TEST FOR CANCER OFFERS LIFE FOR THOUSANDS

A nationwide program emphasizing an annual cytologic test for uterine cancer for all women is urged by Dr. Charles S. Cameron, former Medical and Scientific Director of the American Cancer Society, as a means of reducing the cancer mortality rate.

"Foremost medical opinion is convinced that if every woman in the country had this examination every year, the number of deaths from uterine cervical cancer would be cut by as much as 90 per cent," Dr Cameron writes in a new 25-cent pamphlet, *Cell Examination—New Hope in Cancer*, published today by the Public Affairs Committee, 22 East 38th Street, New York City.

"This would mean an annual saving of 16,000 lives," Dr. David A. Wood, President of the American Cancer Society, 1956-57, declares in the introduction to the pamphlet.

"To make any real dent in mortality figures," Dr. Cameron points out, "a campaign will have to be launched on a national scale that will be carried forward on several fronts at once."

FLAVONOIDS ARE VALUELESS IN DISEASE TREATMENT

As matters now stand, flavonoids are of "little or no value" in the treatment of disease and have no known nutritional uses, according to a report by two American Medical Association councils.

William N. Pearson, Ph.D., of the department of biochemistry of Vanderbilt University School of Medicine, Nashville, Tenn., wrote the report for the A.M.A. Councils on Foods and Nutrition and Drugs.

The report in the Aug. 10 A.M.A. Journal was prompted by "the recent upsurge of interest" in the flavonoids, particularly in the treatment of numerous diseases including the common cold, Pearson said.

Flavonoids are carbon-hydrogen-oxygen compounds that are widely distributed in nature as pigments in flowers, fruits, tree barks and vegetables. Their most important commercial source is citrus rind.

Considerable interest in the possible nutritional significance of these compounds was aroused in the late 1930s by Dr. A. Szent-Gyorgyi and coworkers who isolated "citrin" from citrus fruit peels and reported it to be effective in strengthening capillary and blood vessel walls against break-

age, Pearson said. Such breakage of capillary walls is supposed to occur in a number of diseases.

However, valid tests for measuring the effect of flavonoids on capillary walls have not been devised, Pearson said. An earlier belief that they were a type of vitamin has not been confirmed.

BRAIN GOES 19½ MINUTES WITHOUT OXYGEN

The period the brain can safely go without oxygen has been lengthened from the accepted five minutes to nearly 20—at least in one case, it was reported recently.

Four Illinois researchers told of a 24-year-old man whose brain was oxygen-starved for 19½ minutes during heart surgery, but who recovered with no permanent brain damage.

Damage usually occurs when the brain's blood and oxygen supply is cut off for more than five minutes, they said in the Aug. 10 Journal of the American Medical Association.

They attributed the safe recovery to the fact that the brain was "protected" by the effects of chlorpromazine (a tranquilizing drug given before surgery) and hypothermia (in which the temperature of the body is lowered to slow its functions and allow surgery on the heart).

PACIFIER RETURNING TO POPULARITY

The baby's pacifier, long condemned as unsanitary, tooth-deforming and disease-producing, is making a comeback.

Today many pediatricians and dentists are beginning to look on the pacifier as "at least a partial answer to the vexing problem of how to prevent prolonged thumbsucking and the dental disfigurement it often causes," according to an article by Peter C. Goulding, Chicago, an American Dental Association staff member.

He said bacteriological studies have shown that pacifiers are actually more sanitary than the thumb. In addition, because of their soft texture, pacifiers are far less likely than the thumb to force the teeth out of position.

Perhaps the most persuasive point in favor of pacifiers is the fact that children apparently give them up earlier and with less trouble than they do thumbsucking, Goulding said. One study showed that 28 children spontaneously gave up the pacifier at the average age of 14 months, he said in the August Today's Health, the American Medical Association's popular health magazine.

Most authorities agree that a "basic instinct of sucking" is apparently one of the factors behind the start of thumbsucking. The pacifier helps satisfy this need.

The article quoted Dr. Maury Massler, head of the department of pedodontics at the University of Illinois College of Dentistry, who pointed out that thumbsucking in itself is not bad. But when it is practiced vigorously and during the eruption of the permanent teeth, malformation of the teeth can result.

EYE BLOOD CHANGES SERVE AS RESUSCITATION GUIDE

A once-infallible sign of death might now become a signal of a chance of life as well, a University of Michigan pathologist reported.

Dr. Jack Kevorkian, Pontiac, Mich., said in the Aug. 10 Journal of the American Medical Association that certain changes in the eye—known to occur at death—can also indicate that it is not too late to restart a suddenly stopped heart.

In recent years, sudden cardiac arrest has been overcome by opening the chest and restoring the heartbeat by hand massage and electric shock. This has become an almost common occurrence, mostly during surgery.

But the physician must know whether he still has time for this operation before the brain is permanently damaged by lack of oxygen. Certain changes in the blood supply of the eye's retina can serve as an indicator Dr. Kevorkian said. These changes can easily be seen through the ophthalmoscope, the flashlight-like instrument used by doctors to see inside the eye.

When the heart stops, the flow of blood in the retina's veins (those returning blood to the heart) becomes segmented or interrupted, while the blood in the arteries (those carrying blood from the heart) disappears altogether. The segments of blood in the veins continue to move for several minutes after the heart is stopped.

SYMPOSIUM ON FLUORIDES

The Institute of Industrial Health at the College of Medicine of the University of Cincinnati announces a three-day Symposium on Fluorides to be presented December 9-11, 1957, inclusive. The purpose of this symposium will be to present the most recent information that is available concerning the physiological behavior of the absorption of fluoride.

Discussions will include a brief review of what is known of fluoride metabolism and a considerable amount of data that have hitherto been unreported. The objectives of the symposium are to bring together and to present factual and scientific information on this subject to those who are working in the field of fluoride absorption.

The symposium will be open to physicians and dentists in industry and public health and to other professional persons who are interested in the subject. Attendance will be limited and early application is suggested. The registration fee will be $50.

For further information and application blank write to Secretary, Institute of Industrial Health, Kettering Laboratory, Eden and Bethesda Avenues, Cincinnati 19, Ohio.

ANNUAL MEETING
AMERICAN FRACTURE ASSOCIATION

The American Fracture Association will hold its 18th Annual Meeting at El Paso, Texas, Hotel Cortez, Monday, September 30th, Tuesday and Wednesday, October 1st and 2nd.

The meeting will be preceded and co-ordinated with The University of Texas Postgraduate School of Medicine, El Paso Division which will meet Sunday, September 29th, at the El Paso County Medical Society (Turner) Home, 1301 Montana Street. The University of Texas Program is approved Category I by the American Academy of General Practice. The American Fracture meeting is approved Category II by the American Academy of General Practice.

The program on Sunday, September 29th, at the El Paso County Medical Society (Turner) Home will be on Orthopaedic Surgery. The American Fracture Association Meeting will be limited to fractures entirely.

Registration will begin Sunday for The American Fracture Association at Hotel Cortez.

The American Fracture Association Meeting will start at 8 A. M. each day, Monday, Tuesday and Wednesday. On Monday there will be a registration hour from 8:00 to 9:00 A. M., then the Scientific Program will begin.

BOOK REVIEWS

Medical Department, United States Army, Surgery in World War II. General Surgery, Volume II; Editor in Chief, Colonel John Boyd Coates, Jr., MC. Editor for General Surgery, Michael E. DeBakey, M. D. Associate Editors, W. Philip Giddings, M. D., Elizabeth M. McFetridge, M. A. Washington D. C, Office of The Surgeon General, Department of the Army, 1955. Washington, D. C. Government Printing Office. Price $4.25.

To one who served in the Army Medical Corps in the Mediterranean Theater of Operations during the now not-so-recent "unpleasantness" the feeling of nostalgia engendered by reading this book on general surgery * in that theater comes as a distinct surprise. Names of places and rivers once so familiar, now half forgotten—Venafro, Cassino, the Volturno, the Rapido, the Arno, the Po—these names and many others give rise to a twinge of pain tempered ever so lightly by nostalgia. The reason for the element of pain in a wartime experience is obvious. Why there should be any element of pleasure in one's recollections of that experience is a matter for conjecture.

This book is entirely factual. Yet, without ever once saying so, it is a tribute to the medical officers in the Mediterranean Theater and to what they accomplished. In spite of the confusion and trials of such a cataclysm, the Medical Corps still had the foresight, the physical stamina, and the devotion to their profession to extract from their experience surgical principles which were of incalculable value in the conflict in which they were then engaged, which will be of value in future conflicts, and which are in large measure applicable to civilian medical practice.

That they succeeded in their endeavors is evident in this book, which points up, most strikingly, the improvement in the surgical management of trauma, particularly abdominal trauma, in World War II as compared with World War I. The principles of resuscitation and anesthesia and the management of wounds of the rectum and colon are as timely today as when they were evolved in the campaigns in the Mediterranean Theater between 1942 and 1945. There is no doubt that surgery today is profiting by the lessons learned at the accelerated pace demanded by wartime stress. "It's an ill wind that blows no good" may be a trite adage but its truth is proved in this book.

The volume is well organized. It is divided into three parts, (1) resuscitation, the control of pain, and anesthesia, (2) abdominal injuries, both their general management and the management of wounds of individual organs, and (3) colostomy.

The chapter on resuscitation of the severely wounded can be described as masterful. It is a most valuable piece of reading for any general surgeon, particularly the surgeon interested in abdominal surgery. This section could well serve as a guide for peacetime practice. The importance of a shock ward and the careful and judicious selection of the optimum time for surgery in shock are still lessons to be borne in mind.

The value of whole blood in traumatic shock and the necessity for giving it in adequate amounts are strikingly brought home by the description of how this knowledge was so painfully acquired. The highly original observations on pain in wounded men illustrate the scientific and yet wholly compassionate spirit in which the many problems

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* Volume I of the general surgery series will appear later.

posed by the severely wounded were encountered. These casualties, it was found, did not have nearly as much pain as they were once thought to have, but anxiety was a major psychologic hazard. These observations have important therapeutic connotations which were profitably applied under field conditions and which are applicable in the trauma of civilian life. The recognition of the risks of delayed morphine poisoning and the physiologic basis for its development undoubtedly saved many lives in World War II.

Passing tribute must be paid to the medical officers and enlisted technicians who cooperated to compile the significant data upon which these conclusions were based. They carried out biochemical and other studies and performed postmortem examinations under adverse conditions of weather, poor physical surroundings, and, at times, enemy artillery fire and strafing from planes.

The statistics which necessarily form an integral part of the chapters on abdominal wounds are so presented as to be clear and useful without overpowering the reader, as he might at first fear. They will be of value for future military reference but they are of practical value at this time. One of the surprising facts to emerge from this study was the predominant importance of the number of organs injured (the so-called multiplicity factor) as compared with the influence of the time lag before surgery. Also surprising was the high case fatality rate in wounds of the stomach.

Colostomy was the routine procedure in wounds of the colon and rectum in World War II. The management of colostomies, the chapter which comprises part III of this book, could well serve as a definitive text on the subject. It should be required reading for the surgical resident as well as for the prospective military surgeon. The whole book, in fact, might well be required reading for medical students, internes and residents.

This book is excellently produced, on fine paper, in a most attractive format. The index has been carefully compiled and is very complete.

Any thoughtful reader will appreciate the untold hours of labor that have gone into the preparation of this volume. The editors are to be complimented on the results. Only skill, judgment and devotion to a cause could have accomplished them. It was a remarkable feat to present the principles and practices of general and abdominal surgery in so usable a form; the book could easily have become unwieldy.

Many of the lessons of World War I had to be learned over again in World War II. They were in the official history of the First World War but that history, unfortunately, was not very well known. It is hoped that the circulation of this and other volumes of the medical history of World War II will be so wide that the lessons so unhappily learned in it and so well set forth in this volume will redound to our immediate benefit in any future war.

H. Reichard Kahle, M. D.

Dermatology; by D. M. Pillsbury, W. B. Shelley, and A. M. Kligman, Philadelphia, W. B. Saunders Company, 1956, pp 1331. $20.00.

This is a new text book on dermatology, new in more ways than the fact that it was recently published. It is a large volume of some 1331 pages, yet it omits one of the bulkiest items in most texts, that of bibliography and references. It is different in that there are no lengthy discussions or listings of controversial or time-honored views on etiology and treatment. The knowledge and opinions of the authors are set down in an unusually clear and unencumbered manner which makes for enjoyable reading.

The first two sections of basic principles of skin disease and allergy (174 pages) is a remarkable and delightful condensation of our present knowledge on the subject which few students of dermatology will want to miss reading.

The illustrations (black and white) are well selected and liberally used.

While some dermatologists will not agree with all the views of the authors, all of them will agree that it is refreshing to get the personal viewpoints of such well known authorities as the writers.

Lee D. McLean, M. D.

Bellevue Is My Home; by Salvatore R. Cutolo, M. D. with Arthur and Barbara Gelb, Garden City, N. Y., Doubleday and Co., 1956, Pp. 317, $4.00.

A condensed version of this book was recently published in the Saturday Evening Post Dr. Cutolo, who is Deputy Medical Superintendent of Bellevue Hospital and has been associated with that institution for more than twenty-five years has been principally concerned with presenting to the laity a picture of the operation of a large general hospital. Bellevue is particularly suited for such treatment because its story is long and interesting and illustrates the progress and problems of modern large-scale medicine. The author's affection for his hospital colors his account but does not prevent him from describing some of the difficulties associated with the organization and maintenance of a hospital which holds 2700 patients and has an average daily population of 9700. After the first section of the book which shows Dr. Cutolo at work at Bellevue and tells how he became established there, the successive chapters are devoted to the individual services of the hospital and the spectacular emergencies and unusual diseases with which it has been concerned: The Normandie fire, the New York smallpox cases, and the Empire State Building catastrophe are described in detail. The lay reader will obtain

a reliable picture of progress in surgery, resuscitation and rehabilitation from this book. The physician will enjoy this account of the life and services of a great hospital and should not be disturbed by the simplifications required in a volume designed for the general reader.

MORTON M. ZISKIND, M. D.

Orthopaedic Surgery in European Theater of Operations, Surgery in World War II; by Mather Cleveland, M. D. (Ed.) Washington, D. C., Office of Surgeon General Dept. of the Army. 1956 pp 397. Price $4.00.

This book, which is in a series dealing with the history of the management of war wounds, is especially well organized, beautifully illustrated and concise. Although portions of the book which deal with the organization of the consultant service and the infection of hospitals probably offer little to the practitioner in trauma, other sections of this history are most valuable to any and all who treat the severely injured patient. The sections on hospital facilities, equipment and the numerous well illustrated examples of improvised equipment should prove most valuable to those called on to care for serious skeletal injuries with limited equipment. The description of the management of large numbers of casualties and the management of wounds and compound fractures are sections which are exceptionally outstanding. The illustrations from "Fracture Facts" by Lt. Col. Philip S. Foisie, explaining the need for active exercise in the problem of rehabilitation of injured extremities is both amusing and timely and can well be adapted by any who must deal with the restoration of function in patients who have sustained injury.

Although the detailed study of "Battle Incurred Fractures About the Hip Joint" and "Jeep Injuries of the Hip Joint" are quite impressive, I doubt whether they add much to our knowledge of the management of these lesions which has not already been discussed more adequately and based on a rather larger series than included in this group. This volume should be available to all orthopaedic surgeons and industrial surgeons as well as those interested in developing a medical service for civil defense.

JACK WICKSTROM, M. D.

Dictionary of Dietetics; by Rhoda Ellis, Ph.D., New York, Philosophical Library, 1956, pp. 152, Price $6.00.

This small volume is really more than a dictionary because it gives more explanatory information than is ordinarily found in a dictionary. It is planned for the layman as well as for the dentist, doctor, medical student, student nurse, dietitian, nutritionist, and home economics major. The author is a home economist and a teacher of subjects relating to food and nutrition. Emphasis is placed on the practical application of diet with respect to background food habits and economic status.

MARY LOUISE MARSHALL

PUBLICATIONS RECEIVED

Grune & Stratton, Inc., N. Y.: The Electrocardiogram; Its Interpretation and Clinical Application, by Louis H. Sigler, M. D. (2nd edit.); Systematic Arterial Embolism; Pathogenesis and Prophylaxis, by John Martin Askey, M. D.; Peripheral Circulation in Health and Disease, by Walter Redisch, M. D., and Francisco F. Tangco, M. D.,; Schizophrenia in Psychoanalytic Office Practice, 1956 Symposium; Progress in Psychotherapy, Volume II, Anxiety and Therapy, edited by Jules H. Masserman, M. D., and J. L. Moreno, M. D.; J.A.M.A. Clinical Abstracts of Diagnosis and Treatment, selected by I. Phillips Frohman, M. D.

The C. V. Mosby Co., St. Louis: Gynecologic Surgery and Urology, by Thomas L. Ball, M. D., with foreword by R. Gordon Douglas, M. D.; Pathology, edited by W. A. D. Anderson, M. D.

Philosophical Library, Inc., N. Y.: Foot Troubles, by T. T. Stamm, F.R.C.S.; Surgeons All, by Harvey Graham, M. D.

W. B. Saunders Co., Phila.: Goepp's Medical State Board Questions and Answers, by Harrison F. Flippin, M. D. (9th edit.); Clinical Proctology, by J. Peerman Nesselrod, M. D. (2nd edit.); A Manual of Pharmacology and its Applications to Therapeutics and Toxicology, by Torald Sollmann, M. D. (8th edit.); The Specialties in General Practice, edited by Russell L. Cecil, M. D., and Howard F. Conn, M. D. (2nd edit.); An Atlas of Cardiac Surgery, prepared by Jorge A. Rodriguez, M. D.; Psychosomatic Medicine, by Edward Weiss, M. D., and O. Spurgeon English, M. D. (3rd edit.)

Charles C Thomas, Publisher, Springfield, Ill.: The Pathogenesis of Coronary Occlusion, by A. D. Morgan, M. D.; Blood Pressure Sounds and Their Meanings, by John Erskine Malcolm, Wing Commander, Royal Air Force; The Physiology and Biochemistry of Lactation, by S. J. Folley, Ph.D.; Plastic Arterial Grafts, by W. Sterling Edwards, M. D.; Chemistry of Erythrocytes, by Hans Behrendt, M. D.; Psychiatry in Theory and Practice, by Beulah Chamberlain Bosselman, M. D.; Degenerative Changes in the Sternoclavicular and Acromioclavicular Joints in Various Decades, by Anthony F. DePalma, M. D.; Kaposi's Sarcoma, by Samuel M. Bluefarb, M. D.; Therapeutic Exercises for the Treatment of the Neurologically Disabled, by Harold J. Brenner, M. S.; The Placement of Adoptive Children, by J. Richard Wittenborn; Essentials of Fluid Balance, by D. A. K. Black, M. D.